POCKET BOOK OF PHYSICAL DIAGNOSIS

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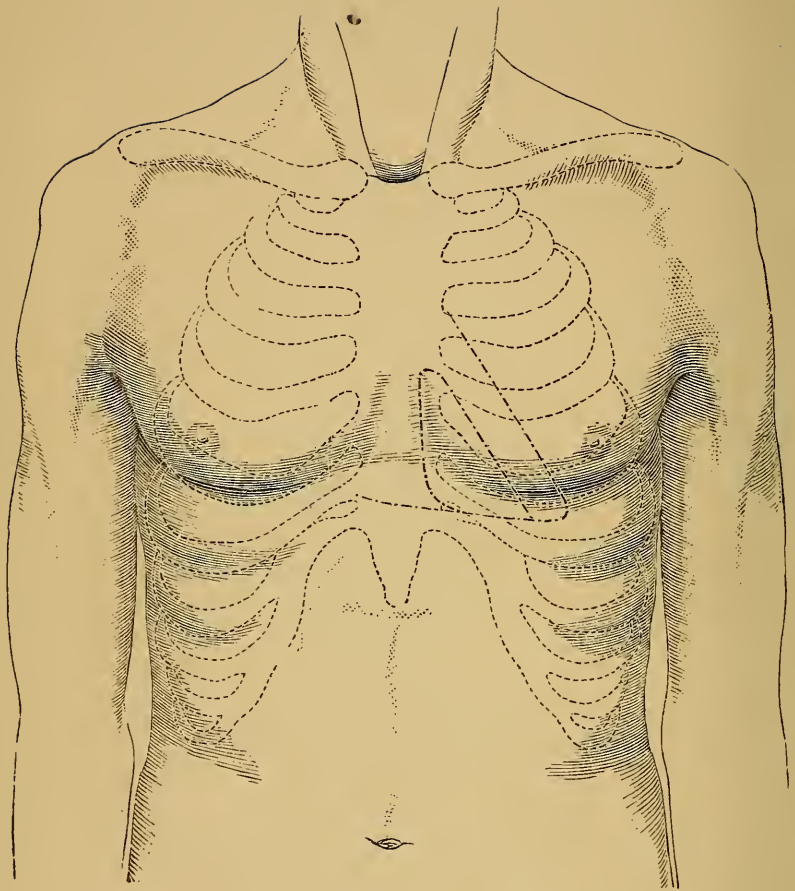

Surface of Normal Chest.
Outlines of Cardiac Dullness to the left of the Sternum.
The spaces enclosed in dotted lines represent the inter-spaces.

A

POCKET BOOK

OF

PHYSICAL DIAGNOSIS,

OF THE

DISEASES OF THE HEART AND LUNGS.

FOR

THE STUDENT AND PHYSICIAN.

BY

DR. EDWARD T. BRUEN,

DEMONSTRATOR OF CLINICAL MEDICINE IN THE UNIVERSITY OF PENNSYLVANIA, AND ASSISTANT PHYSICIAN TO UNIVERSITY HOSPITAL; ONE OF THE PHYSICIANS TO THE PHILADELPHIA HOSPITAL AND DISPENSARY OF THE CHILDREN'S HOSPITAL; LECTURER ON PATHOLOGY IN THE WOMAN'S MEDICAL COLLEGE OF PHILADELPHIA, ETC.

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PREFACE TO SECOND EDITION.

When the processes of pathological action have produced structural changes in the tissues forming the various organs and framework of the body, the science of physics, which takes cognizance of the changeful properties of matter, enables us to institute methods of examination to which the term physical diagnosis forms a clear and appropriate designation. It should be borne in mind that no single fact determined by the methods of physical diagnosis has special pathological significance, but simply indicates certain definite physical conditions of the organ under examination. The purely objective physical signs are so closely intertwined with the general symptoms of disease, that any study of diagnosis is partial which does not recognize this fact. Both physical signs and general symptoms must be in their turn considered in connection with a thoughtful analysis of the processes of morbid anatomy, because symptomatology is the study of the expression of pathological changes.

The subject is treated in as practical a manner as possible, without discussion of questions of historical or theoretical interest, and without laying special claim to originality of matter.

1531 Chestnut St., Philadelphia,
September 1st, 1883.

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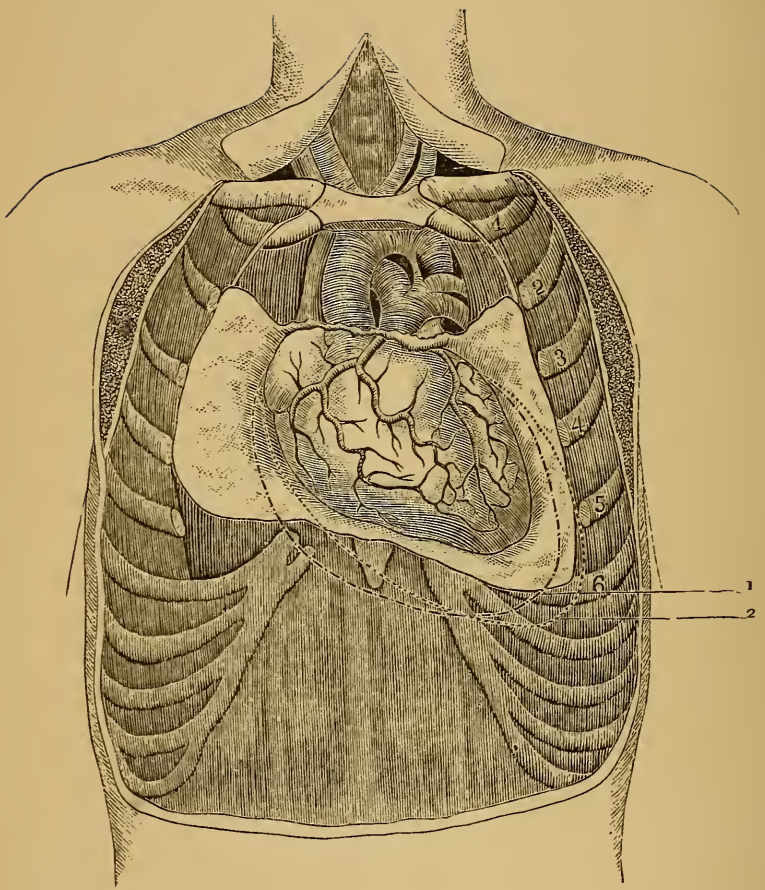


1 Area of Hypertrophy of Right or Left Ventricles.
2 Area of Dilatation.

INTRODUCTION.

The general appearances of the healthy adult chest are sufficiently familiar to need little description. In the male the shoulders should be moderately broad, square and level. In shape, the chest is compressed antero-posteriorly, the back being comparatively flat, while anteriorly the walls of the chest present an ample lateral curve, and slope gradually upward to the clavicles. The nipples are situated in the fourth interspace, but their position is variable. In the female the shoulders are more rounded and sloping, the sides of the chest taper downward toward the waist, while in front the large accumulation of adipose tissue in the sub-clavicular regions and the prominent mammary glands conceal the form of the bony thorax.

When the soft parts are removed the bony case appears in both sexes as a truncated conoidal form, flattened antero-posteriorly, with a broad base, and a round apex, the lower end of the sternum being twice as far away from the spinal column as the upper end. The greatest circumference is just below the middle. The female chest is less deep than the male. The transverse section is elliptical. The trachea enters the upper aperture of the chest in the median line, and descends opposite the third dorsal vertebra, where it divides into two bronchi, one for each lung. The right bronchus, which is wider, shorter and more horizontal in direction than the left, is about one inch in length, and enters the right lung opposite the fourth dorsal vertebra. The left bronchus is nearly two inches long, and passes more obliquely downward, entering the root of the left lung, near

the fifth dorsal vertebra. The lungs are attached, a little above the middle of their inner surface, to the trachea and heart, by their roots, which contain, in addition to the bronchus, a branch of the pulmonary artery, pulmonary veins, bronchial arteries and veins, the pulmonary plexus of nerves, also lymphatics, and bronchial glands. The apex of the lung forms a tapering cone extending from one to one and a half inches above the level of the first rib, slightly higher on the right side. The base of the lung is broad and concave, is opposed to the convex surface of the diaphragm; its circumference is thin and extends anteriorly down to the lower border of the sixth rib, laterally to the seventh rib on the right side, and a little lower on the left side, and posteriorly down to the eleventh rib. The posterior border of each lung lies in the deep groove behind the spinal column; it is thick, rounded, and covers the root of the lung. The anterior borders are thin and sharp; they extend forward into the mediastinal space, so as to come into contact with each other, as far down as the fourth rib; below that level the border of the right lung in the median line of the sternum continues to encroach upon the sternal region, while on the left side it retreats from the sternum, leaving a considerable portion of the pericardium uncovered, in an irregularly sloping direction.

The pericardium is conical in shape; its base is attached to the central tendon of the diaphragm, extending a little further to the left than to the right side; its apex is directed upward, and surrounds the great vessels just beyond their origin from the base of the heart. It is situated behind the sternum and the cartilages of the third, fourth, fifth, sixth, and seventh ribs, on the left side, but it is in part covered by the anterior margins of the lungs. Its upper limit is about the lower edge of the second rib. The portion of the sac which is uncovered by the lungs is widely triangular in shape, and extends from the fourth interspace down to the

sixth rib, and from the median line of the sternum out to within one-half inch of the line of the left nipple.

The heart lies obliquely in this sac, held by its attachment to the great vessels which spring from its base. This broad detached end is directed upward and backward to the right, and corresponds to the interval between the fifth and eighth dorsal vertebræ. On the anterior surface of the chest its upper border corresponds with a line drawn on a level with the upper border of the third costal cartilage. The lower border of the heart corresponds to a line drawn from the xiphoid cartilage (right costo-xiphoid articulation) along the upper border of sixth rib to the position of the apex, which is situated in the fifth interspace, an inch inside the line of the left nipple, or two and a half inches from the median line of the sternum. The heart is placed beneath the lower two-thirds of the sternum, and projects a little beyond the right sternal border.

In percussion of the præcordia we have two rudely triangular areas of resonance; one of impaired resonance, one of flatness. The area of flatness is that district over which the edges of the lungs do not meet. This area is bounded at the mid-sternal line by level of the fourth left costo-sternal articulation. Prolong a line from this point to the apex of the heart. Complete the right-angled triangle by projecting a line from the apex beat to the mid-sternal line once more. The other boundaries correspond to the area of impaired resonance. The two triangles vary slightly with the position of the patient, either sitting or standing. Percuss with the fingers held vertically, commencing a little to the right of the sternum, until dullness is recognized. Continue the percussion until beyond the apex pulmonary resonance is again reached. Then place the fingers parallel with the ribs to the left of the apex, and percuss gradually inward, interspace by interspace, till the sound changes from resonance to impaired

resonance, to dullness. Afterward, by placing the finger used as a plexímeter obliquely pointing towards the sternum, commencing at the apex, the previous percussion can be checked. The upper limit of dullness can be defined by percussion from above downward, the fingers parallel to the ribs. The lower limit is not so easily defined, since the cardiac dullness blends with that of the left lobe of the liver, and the dullness of the two organs can scarcely be discriminated. A line of dullness can, however, be defined between the apex on the left and the commencement of liver dullness on the right. The position of the areas of cardiac dullness is much influenced by the degree of distention of the intestinal tube. If the diaphragm is elevated, the heart is carried upward.

The aorta and pulmonary artery are the only vessels connected with the base of the heart which furnish physical signs of much importance. The aorta commences opposite the middle of the sternum, on a level with its junction to the third costal cartilage; it passes upward to the right for a distance of about two inches, almost in the direction of the heart's axis, as high as the upper border of the second right costal cartilage, and about a quarter of an inch behind the posterior surface of the sternum. It then passes from right to left, and from before backward to the left side of the second dorsal vertebra. In this part of its course its upper border is usually about one inch below the upper margin of the sternum. After reaching the third dorsal vertebra the aorta descends in a straight course, at first lying on the left side of the spine, but approaching the median line as it descends, until it passes through the aortic opening in the diaphragm.

The pulmonary artery is a short, wide vessel, about two inches long, arising from the left side of the base of the right ventricle; it crosses in front of the ascending aorta at the level of the third costal cartilage, and near the left margin of the sternum. It extends upward, backward and slightly

to the left as far as the under surface of the arch of the aorta, when it divides into the right and left pulmonary arteries. The right branch is somewhat larger than the left; it runs horizontally outward, behind the ascending aorta and superior vena cava, to the root of the right lung. The left branch passes horizontally in front of the descending aorta and left bronchus, to the root of the left lung.

It is important, also, to be familiar with the relative position of the viscera which occupy the upper portion of the abdomen, since by their encroachments upon the area of the thorax they materially modify the results of physical examination.

The liver lies transversely, its right lobe occupying the right hypochondriac region, while the left lobe reaches across about two inches beyond the median line. Its upper border is at the level of the fifth intercostal space; but, owing to the convexity of the upper surface, is separated some distance from the anterior thoracic wall. It is capped, as it were, by the concave base of the lower right pulmonary lobe. It comes in contact with the chest wall (the diaphragm alone intervening) about the level of the sixth rib. The lower margin of the liver usually corresponds with the free border of the ribs. In the line of the axilla, the upper margin of the liver reaches to the seventh rib, and extends beneath the posterior surface of the chest to the lower margin of the tenth rib. On account of congenital variations in the size of the liver and its possible elevation by conditions of the abdominal cavity—tympanitic distention, dropsy, abdominal tumors, etc., it is well to form an idea of the dimensions of the liver expressed in figures. Anteriorly, the region of dullness and flatness includes a distance of about three inches in the line of the right nipple. Laterally, in the axilla, it includes a space measured by about four inches. Posteriorly, the measurement is from two to two and a half inches.

Percussion of the liver should be made both in the erect and recumbent positions. It is much easier to define the transition from dullness to the tympany of the abdominal region when the patient assumes the recumbent posture, since the abdominal walls become more flexible. The thick abdominal walls possessed by some effectually preclude an easy recognition of the tympanitic resonance, and might lead the incautious to infer enlargement of the liver when none exists.

In percussion, place the finger used as a pleximeter parallel with the ribs, commencing, we will say, at the third interspace. Percuss downward until absolute flatness is noted even in forcible percussion; then percuss more gently upward, noting, meanwhile that the resonance becomes more and more clear as one ascends the chest, until unmixed pulmonary resonance is determined. Unless there is disease of the pleural cavity, or the base of the right lung, the boundaries of the liver can easily be defined. Frerichs has given us a rule which is very useful,—"the liver dullness can always be altered by a full inspiration; but if there is a pleural effusion, or chronic pneumonia, the level of impaired resonance or dullness is not changed during respiration."

The spleen occupies the upper portion of the left hypochondriac region. It is about four inches long, reaching from the free border of the ribs up to the ninth rib. It is about three inches wide, its anterior border lying in contact with the stomach and colon. Its convex surface is closely opposed to the concavity of the diaphragm, so that it influences the results of percussion in this region over nearly the entire extent of the organ. If the stomach contain much food, or if it or the intestines be distended with gas, it is difficult to detect the splenic dullness. Percuss in the mid-axillary line, and at the ninth or tenth rib the resonance becomes dull, and the dullness should extend about three

and a half inches. By percussing from the median line to a point between the lines which bound the inferior and superior margins, we notice where stomach tympany ceases and dullness begins. When the spleen cannot be detected by percussion, we may infer it to be small, unless the stomach and intestines are not much distended with gas.

The stomach varies widely in its dimensions and relations, under different circumstances, in the same individual. When moderately distended with gas it is comparatively easy to determine its boundaries and position. The cardiac orifice corresponds to the inner extremity of the seventh left rib, while the pylorus is found near a line drawn from the right nipple to the umbilicus, a little below the lower border of the liver.

The left or posterior border of the organ is bounded by the spleen; the lower border is usually nearly on a line with the umbilicus. The upper border is in contact with the under surface of the diaphragm and liver.

The colon is divided into four parts, the ascending, descending, and the sigmoid flexure. For our present purposes we must bear in mind the distribution of three parts. The cæcum is situated in the right iliac fossa, immediately behind the abdominal wall; the ascending colon is smaller, and passes upward to the under surface of the liver, on the right of the gall bladder, where it bends abruptly to the left, and in this part of its course is spoken of as the transverse colon. The ascending colon is in relation in front with the convolution of the ileum and the abdominal walls; behind, it lies on the quadratus lumborem muscle and right kidney. The transverse colon is in relation, by its upper surface, with the liver and the gall bladder, the greater curvature of the stomach, and the lower end of the spleen; by its under surface with the small intestines; by its anterior surface with the anterior layers of the great omentum, and the abdominal

parietes. The descending is more deeply placed than the ascending colon, and is smaller in calibre. Its relations are similar to those of the ascending colon.

The loops of the small intestines are surrounded, as it were, by the distribution of the colon. Normally they occupy a central position in the abdominal cavity. They are, however, loosely confined by thin mesenteric attachment, a fact of no little importance to the diagnostician, since they can be displaced in various directions by accumulation of fluid, or tumors occurring in the abdominal cavity. Percussion of the abdominal region should be made when the abdominal parietes are relaxed, if this be practicable, since the pitch pertaining to each of these cavities can be more readily distinguished. The percussion note over the stomach, colon and small intestine are alike in quality, viz., tympanitic, but vary in pitch.

The tympanitic resonance of the stomach is much lower in pitch than the resonance over the colon, and the resonance of the colon is lower-pitched than the tubular or high-pitched tympanitic resonance of the small intestine. The reasons for this variation are elsewhere stated.

The kidneys are situated in the lumbar regions, in the space corresponding to the two lower dorsal and the two upper lumbar vertebræ; the right is a little lower than the left. Superficially, they extend from near the eleventh rib to the crest of the ilium. The right is bounded above by the posterior and inferior portion of the right lobe of the liver; below, by the cæcum; anteriorly, by the ascending colon, and posteriorly by the diaphragm and quadratus lumborem muscles. The left is bounded above by the spleen; anteriorly and inferiorly by the colon, and posteriorly by the diaphragm and quadratus lumborem muscle. These organs are so deeply situated, and so surrounded by muscular structures, that unless they are enlarged, it is difficult to demon-

strate their presence satisfactorily by percussion. Having thus briefly indicated the normal anatomical position of the thoracic organs and of the abdominal viscera, whose relations with them influence the result of physical explorations, we must also indicate a few of the more important of the vast number of changes within the abdominal cavity which influence physical diagnosis.

Influence of some Abnormal Abdominal Conditions upon the Thoracic Viscera.—Distention of any part of the intestinal tube with gas will tend to elevate the diaphragm, materially interfere with respiration, and displace the heart, liver, or spleen. The stomach is capable of enormous distention by gas, especially whenever its pyloric orifice is seriously stenosed. The cardiac extremity can arise so high as the sixth or seventh rib in the left axilla. A ready means of identification is practiced by causing the patient to drink slowly a glass of water—the metallic tinkle of the fluid as it drops into the viscus is very perceptible. This method is very useful to differentiate distention of the stomach from pneumothorax, or emphysema of the left side.*

Abdominal distention is capable of abrogating the diaphragmatic movements sufficiently to seriously modify the respiratory murmur. The pitch, quality, and rhythm of the murmur can all be influenced; but principally the last two attributes. The lungs may be so compressed that the quality of the respiratory murmur may become concentrated, harsh or broncho-vesicular, and the pitch elevated, especially in the upper anterior and posterior portions of the chest, and the rhythm can be changed so that we hear only the inspiratory movement of respiration; or the breathing may be interrupted in rhythm. In other instances, the quality becomes feeble, and the pitch very low. The displacements of the heart may embarrass its revolution, and cause modifications of

* *See* section on Pneumothorax, and on Emphysema.

rhythm, which it would be tedious to describe in detail. Even murmurs can be temporarily suppressed. Friction sounds, both pericardial or pleural, may temporarily vanish, by reason of extreme gaseous distention of the abdomen.

Dyspnœa is naturally a most important and frequent symptom, since the aeration of the blood cannot be effectively performed. Large abdominal effusions or tumors in the abdominal cavity are mechanically capable of interfering with respiratory action. In the male the inconvenience is observed more markedly in the respiratory organs than in the female, since the former are less capable of exaggerated superior costal respiration. Acute or chronic lesions of the peritoneal membrane are also attended with deficient play of the diaphragm, and more or less modification of the respiratory movements.

PHYSICAL DIAGNOSIS

PART I.

OF THE LUNGS.

CHAPTER I.

PERCUSSION.

The most useful methods of physical examination are by means of percussion and auscultation. The other methods include palpation, inspection and mensuration, but are auxiliary to the first named. In percussion, the position of the patient should be standing, if the most accurate results are to be gained; next to this the sitting posture, with the arms so placed as to render moderately tense the tissues covering the chest. If the abdominal region is to be studied the percussion may be made while the patient is lying on the back, or standing, so that the resonance obtained while the planes of the tissues are in varying states of tension, may be attentively studied and compared. The best instruments for performing the act of percussion are the hands; the fingers of one hand being used as a plexor, those of the other as a pleximeter. The finger used as the pleximeter should be the fore-finger, but the middle or ring-fingers should be used when it is deemed desirable to percuss in succession several parallel planes of the chest. The fingers should be applied so as to firmly compress the various layers of tissue forming the walls of the chest or abdomen into one compact layer; the pitch of percussion is much modified by neglect of this practice, and

mistakes are rendered possible, especially in the more disseminated and moderate pathological changes. Another value of the digital pleximeter is that by it we are enabled to appreciate the amount of resilience or elasticity of the tissues percussed. Piorry was wont to remark that he felt the modifications of percussion resonance.

As a plexor, we employ one or two fingers of one hand, which should be placed in such relation to each other that the tips are of equal length. Percussion may at times be made with one finger only; this is especially the case when it is desired to elicit but a moderately intense sound, but if resonance is to be developed from deep planes of tissue, then both fingers should constitute the hammer. The stroke itself should be delivered from the wrist, and by the impulse of the hand alone. The secret of percussion, as in piano playing, is to encourage flexibility of the wrist, and as the execution of the pianist lies in the touch, which is secondary to a trained freedom of wrist movement, so touch in percussion depends on a similar training of the wrist, not on manual force. The knack of percussion lies in the ability to develop full vibration in resonant tissues, not in the forcefulness of the blow.

The essential properties of resonance, viz: quality or tone, and pitch, are much affected by the method of percussion, and often depend quite as much on the execution as on the state of the tissues. For example, the pulses of air should follow rhythmically on one another, and should be repeated at regular intervals and with regular rapidity. It has been found by experiment that the number of waves must not be fewer than forty in a minute; otherwise they do not blend into a tone; vice versa, tone is influenced by their too great rapidity. The pitch of a sound depends on the swiftness with which the periodic waves follow each other, and in the number of shocks following each other in a given space of time; the swifter the succession of both, the higher the pitch.

It is for this reason that faulty percussion is capable of developing sounds which lead to erroneous results. Percussion, with both rapid and slowly repeated shocks must be compared; those given with medium rapidity are usually the most satisfactory. In the selection of a site on the chest for percussion, if possible, choose the interspaces in the chest, rather than the bony ridges; the only caution we advise is that care must be taken to compare site with site on the opposing sides of the chest. It is advisable, when possible, to percuss placing the pleximeter parallel with the axis of the ribs; exceptionally, it may be necessary to place the pleximeter perpendicularly to the axis of the body.

Definition of Resonance. We make use of the term quality or tone of resonance in order to enable us to refer sounds to classes and thus form a standard for comparison. We recognize the difference in quality or tone of stringed as compared with wind instruments, or the sound emitted from striking a glass or a metal object. Pitch, however, is the key or note evolved when the quality is developed. Pitch depends upon the number and rapidity of the vibrations in any given resonance. For example, the pitch of a stringed instrument depends upon the tension of its strings; that of a wind instrument upon the calibre of its pipes. In the first example the vibrations of the strings is more or less rapid, and the pitch correspondingly high or low; in the wind instrument, which is more suitably compared with the human chest, the pulses of air follow each other with varying degrees of swiftness, according to the amplitude of the pipes.

The word resonance as applied to the lungs, is a cardinal term indicative of reverberation, to be contrasted with deadness, or such complete absence of musical quality that a sound comes to be termed flat or toneless. Therefore, pulmonary resonance does not consist, fundamentally, in volume or loudness; this is an incidental attribute depending largely

on the forcefulness of the motor impulse generating it, and on the condition of the tissues encasing the lungs; and on the same principle, intensity is not a factor in the integral composition of resonance. The duration of resonance is dependent on the intensity of sound, and must also be laid aside in the inquiry as to the entity of resonance.

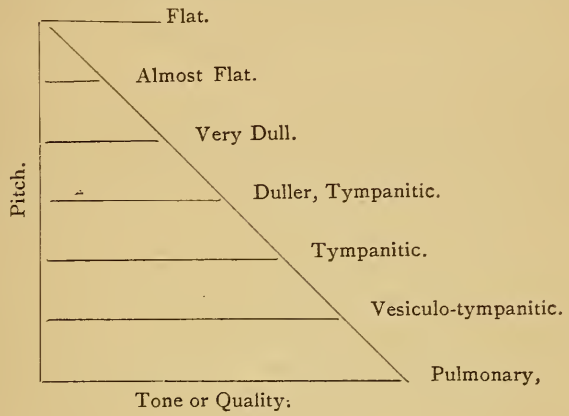
Avoiding familiar anatomical details of the structure of the lungs, the respiratory apparatus for the purposes of the clinical diagnostician, consists of the trachea and bronchial tubes, which form a tree the branches terminating in the finest bronchioles. Surrounding these are spread out a multitude of sacculations containing air, that is, the vesicles and lobules of the lungs.

It is chiefly important to recognize that the vesicular pulmonary tissue is elastic and confines the air under a moderate condition of tension. The bronchial tissue is less elastic, the contained air is confined within a more limited space, and its state of tension is more decided. When we consider the stomach and intestinal canal we find it to be an air-containing tube, of varying diameter, and its walls sustaining varying degrees of distention.

Basing our observations upon the above statements, we easily see that the quality or tone and pitch of the resonance of the digestive tube depends upon the varying size and tension of its walls, due to their distention by gases. But a few moments' practice will convince any one that over the stomach, if fully distended with gas, the sound is much more acute—high pitched—as compared with the graver—low pitched—note of the less distended viscus, and this irrespective of the size of the organ. A similar statement is true concerning the small intestine and colon. The tension and elasticity of the walls of the digestive tube indicate that the vibrations of the air, if percussion is made, will be shorter in proportion as the tension is increased and the elasticity diminished, and the

pitch will be higher; just the opposite will occur if the reverse condition obtains. If percussion is practiced over a solid viscus, such as the liver, it must yield a toneless sound, that, in common parlance would be termed flat or non-resonant, since its structure is not air containing.

To represent this diagrammatically let me erect this figure of a triangle, which can be filled in as we proceed:—



Normal Pulmonary Resonance. To understand the term pulmonary resonance, which must become a typical sound, let us fancy resonance from air contained in the vesicular and bronchial tissues of the lungs, versus resonance from air distributed in the digestive tube; have we not at once the mental picture of a sound which is less tympanitic in quality, because less hollow in quality. It is less hollow because the air is distributed in smaller amounts in any given district; more low pitched, because the wave of vibration of the air after impulse is longer, since the tissues are more elastic and the air is under a lower state of tension. But the respiratory acts must modify percussion resonance. Consider the effect of a full-forced inspiration upon the normal pulmonary structures. The volume of air contained within the chest and the capacity of the air sacs is increased; the tension of the walls

of the vesicular pulmonary tissue is notably exaggerated, while the elasticity is impaired. As the outcome the percussion resonance departs from the typical and approaches the tympanitic; but since it partakes of the qualities both of pulmonary resonance and the tympanitic, it has been most justly termed vesiculo-tympanitic, or a sound of more high pitch than good pulmonary resonance, more tympanitic in quality, but still retaining sufficiently the qualities of normal vesicular resonance to deserve its christening as vesiculo-tympanitic resonance.

Exaggerated Resonance. Exaggerated resonance is a term employed by some, but is practically synonymous with vesiculo-tympanitic resonance, from which it differs only in having a less hollow quality, and not quite so high a pitch. Examples of this kind of resonance occur in cases of supplemental action of a single lung from disablement of its fellow by the filling of its pleural cavity with fluid, or by the various forms of pathological process to be subsequently described. It is also, when general over both lungs, the evidence of substantive emphysema.

Influence of Tissues on Resonance. With these results of percussion, the flat, the tympanitic, the normal pulmonary resonance, and its modification, the vesiculo-tympanitic, the next step must be to consider the distribution of resonance in the physiological chest, and the influence of special conditions of the chest walls. The ability to map out the organs contained within the chest is one of the final steps in the study of percussion. The most typical pulmonary resonance can be elicited over the upper anterior portion of the chest, above the third rib; in the axillary regions; and posteriorly below the angles of the scapulæ; the best resonance being located in the regions in the order as named.

In the other regions of the thorax, it is evident that the layer of bones and muscles must seriously impair the clear-

ness of resonance; for instance, in the regions occupied by the scapulæ and the thick muscles of the back. It is also modified in those cases in which the distribution of adipose tissue is unusually large. The above conditions modify chiefly the volume or intensity of the sound, but the quality may be dull or almost flat, and the pitch correspondingly elevated. In individuals who are very thin by constitutional habit, and in others who have become thin through the atrophic changes in the tissues incidental to disease or advanced age, the resonance is often of the vesiculo-tympanitic type.

It occasionally happens, in the physiological chest, that the percussion resonance over the right apex verges toward the vesiculo-tympanitic, or, at least, is more high pitched than on the left side; it is less significant of pathological condition than a similar modification over the left apex.

In childhood the bony thoracic walls are slight, and the integument and muscles are distributed in thinner layers. On this account we are able to develop more easily vibrations in the air contained in the lungs, because the percussion impulse impinges more immediately upon the pulmonary substance. In children, also, the bronchial tissue predominates, consequently the pulmonary tension is increased and the pulses of air must be shorter, therefore the exaggerated or vesiculo-tympanitic resonance is physiological in children.

Respiratory Percussion is a method which has been described by Dr. Da Costa as sometimes very useful. It consists in practicing percussion, either at the moment of full inspiration, while the person examined holds the breath, or at the end of forced expiration.

Over the apices, percussion at the end of full inspiration yields a sound of higher pitch and vesiculo-tympanitic (fuller vesicular) quality. The usual disparity between the right and left sides is preserved, the pitch on the left side remaining

lower than the right. Broadly speaking, this effect can be noted more or less positively in all the regions of the chest.

A held expiration diminishes the resonance, but in the writer's experience respiratory percussion is most useful during the inspiratory movement.

Another valuable method yielding useful data as to the state of the pulmonary tissue of an entire side, is percussion over the clavicle. Its results, in quality and pitch, often supersede the necessity of further investigation—a point of importance when it becomes desirable to shorten an examination.

Auscultatory Percussion is performed after pressing the stethoscope firmly against the interspaces, and then practicing percussion. This method is very useful in examining cases of suspected pneumothorax, and is sometimes an assistance in mapping out organs.

Finally in describing the locations of resonance, the ribs constitute the appropriate landmark, with the median line of the sternum as a fixed dividing line between the two sides of the chest. This is of especial importance when we attempt to record for others the limits of abnormal resonance; and it will prevent mistakes to define the record by inches.

It is a general principle presiding over physical diagnosis, that each result is based on the comparative examination of the two sides of the chest in each individual case. This is especially important in connection with auscultation and percussion. Questions of quality and pitch being relative terms, each case must be an independent study and a law unto itself.

CHAPTER II.

AUSCULTATION, PALPATION, MENSURATION, INSPECTION.

By auscultation we study the respiratory murmur, or the passage of air through the bronchial tree, from the trachea to the finest of the terminal bronchioles, until it is lost in the vesicular structure. Physiologically speaking, it is a murmur separated into two movements of equal length, the one occurring during the inspiratory, the other during the expiratory act. Clinically speaking, it is a murmur with a movement of greatest duration during the inspiratory period, which may be diagrammatically indicated by the numeral 5, a period of pause, indicated by the figure 1¾, and the period of expiration, represented by figures 3¼. The expiratory murmur is longer over some regions of the chest than others; for instance, in those regions most liberally supplied with bronchial tissue, and in persons who possess some of the physical conditions which combine to yield a vesiculo-tympanitic percussion. The respiratory murmur presents for our study the movements of inspiration, a pause, then expiration. The sequence of these acts constitutes what is called *rhythm* by writers on diagnosis. The pause occurs during the period of vesicular collapse, while the air is passing from the vesicles into the bronchial tube, but so noiselessly as to be inaudible, so that we hear only the exit of the air from the bronchial tubes, and call it the expiratory murmur, whereas, it is but a part of it.

Like percussion, the chief attributes of the respiratory murmur are quality and pitch. *Intensity* or *volume* of murmur has only a relative diagnostic importance, since it is wholly dependent on the amount of air which can enter and find exit from the lungs, while the breathing in quiet respiration

may be so shallow and feeble that its elements may be much masked. When the respiration is deeper, we classify it chiefly by its quality and pitch.

The *duration* of the respiratory murmur varies in accordance with the rhythm. Deviation in the duration of the respiratory acts is valuable, when associated with changes of quality and pitch, to mark certain pathological conditions.

In studying the respiratory murmur we award the place of first importance to quality, then pitch, relegating rhythm and volume to positions of subordinate value.

There are three *types* of *respiratory murmur* in the physiological chest, viz.: the tubular, heard through a stethoscope placed over the trachea; the bronchial, heard most characteristically between the scapulæ, the region principally occupied by bronchial tissues, and most distinctly audible in individuals who are thin; the vesicular, most audible at the anterior and upper portions of the chest, in the axillæ and in the infra-scapular regions. The tubular type is a murmur of a very concentrated or reed-like quality, hence its name; and the pitch is necessarily high, since the tube in which the sound is generated is not a large one. Essentially the concentration and high pitch are the most important factors of this sound. The bronchial type is a natural derivative from the tubular; it differs only in being less concentrated, less high pitched, although its tubular quality remains. It is simply named bronchial, from the tissue in which it is produced; with equal propriety it might be called less pronounced tubular breathing. The diffusion of the sound and the lower pitch of bronchial as compared with tubular breathing are to be explained by the situation of the bronchial tubes, embedded in a vesicular tissue which acts as padding might around a series of reeds of similar size.

The *vesicular breathing, or normal respiratory murmur*, consists in a further modification of this type: the air passing

into the vesicular tissue expands it with a rustling sound, which is diffused, because the elastic vesicular tissue constitutes the bulk of the lungs; it is low pitched, because the cavities receiving the air, though limited in size, are very numerous. This murmur, termed by writers breezy, is typical to express the lowest pitched and the least concentrated sound which the student of physical diagnosis has to observe.

The first modification of the quality of the respiratory murmur is termed the broncho-vesicular. It consists in a modified form of bronchial breathing, in which the bronchial element is masked by an exaggeration of the vesicular portion of the respiratory murmur. Consequently it is a higher pitched sound than normal vesicular breathing but not so high pitched or so concentrated a sound as bronchial breathing. Further, since the amount of air entering the chest is increased, the volume or intensity of the respiratory murmur is increased when compared with a physiological standard. Broncho-vesicular breathing occurs when a lung has undertaken supplemental activity, it is also very marked in children, since the bronchial tissue exists in excess of the vesicular; conditions which render the pitch higher and the quality harsher. When the chest walls are thin the breathing becomes broncho-vesicular. Broncho-vesicular breathing, in addition to the above instances, is heard in certain forms of emphysema, especially the atrophic, and in special stages of consolidation, to be hereafter described.

Broncho-vesicular respiration is equivalent to the term exaggerated respiratory murmur, although by some writers a distinction has been made, broncho-vesicular respiration indicating for them slight consolidation of the pulmonary tissues. If broncho-vesicular breathing is associated with any process of condensation of vesicular tissue, the concentration of quality is more distinct and the pitch of expiration grows higher as the

process of condensation progresses, in contrast with the diffuse quality and more low-pitched sound when consolidation is absent; while the normal respiratory murmur is still more diffused in quality, of less volume and lower pitch.

The study of *rhythm* relates to the appreciation of the time of the respiratory revolution and its deviations from the standard. The physiological rhythmic development of any of the respiratory sounds depends chiefly on the amount of air introduced into the chest; so that in healthy persons partial inflation will develop a respiratory murmur of atypical rhythm, because the expiration will be shortened. Disease presents many such examples: in early stages of phthisis the respiratory murmur is pronounced; as the process advances less air is inspired, and feeble respiratory murmur, especially on expiration, often becomes an important evidence of the change.

Reversely considered, the preponderance of bronchial tissue in a lung, or the nearness of the bronchial tissue to the ear of the listener, will combine to render the expiration more and more distinct, until it may become equal to the inspiratory murmur. It is evident that the rhythm of respiratory murmur depends, to a great extent, on the relation of the bronchial to the vesicular structure, and the thickness of the thoracic walls, and the amount of air respired. The study of rhythm is of great importance in recognizing conditions of consolidation and other pathological changes.

Normal Distribution of Respiratory Murmur. Inspiration is heard as a more prolonged murmur than expiration, over the apices and in the axillary regions; in the infrascapular the expiratory sound is sometimes less distinct than in the first named situations. Between the scapulæ the bronchial tissue predominates, and the respiratory murmur during the two movements of respiration is more nearly equal.

Exceptions. Occasionally the quality, pitch and rhythm of the respiratory murmur are altered, so that over the right apex the pitch is higher and the quality less vesicular than over the left; the rhythm also changes, the expiration being somewhat more prolonged. No very adequate explanations have been proposed for these physical signs. It has been thought more common in right-handed persons, but a better working rule will be to carefully investigate, by all the methods of physical diagnosis, whenever any pronounced difference between the two sides of the chest can be detected. Note if the habit of using the muscles of the side is influenced by occupation or business, and decide in favor of physiological variation only after a strict analysis, by exclusion of any pathological process.

Method of Auscultation. In practicing auscultation it is usually desirable to examine a considerable area of the chest. This is best accomplished by applying the unaided ear to the thorax, which has previously been stripped to one or two coverings, over which the examiner can place a smooth, soft towel. With both men and women, when accuracy of investigation is essential, this rule must be absolute. Silk or merino underwear, unstarched linen or muslin garments, may be worn without interfering with the examination. But in all subacute or chronic cases, the satisfaction to the practitioner of accurate knowledge, and the comfort to the patient of an assured opinion, are so obvious, that the trouble required for an examination will readily be yielded. The science of physical diagnosis has received its worst blows from the hands of hurried and partial, rather than unskilled investigations.

But, at times, it is desirable to auscult a limited area, or acute disease may make changes of attitude necessary for examination by the ear impossible. For this reason it is necessary also to become familiar with the use of some reli-

able stethoscope. None is better than that made of thin bell-metal—now to be procured of Mr. Gemrig, of this city. Its weight is one ounce, and as a perfect conducting medium, it is unsurpassed by any with which I am acquainted. Certain other stethoscopes are vended which are not so desirable. They may be recognized by the thickness of the barrel of the instrument, the thickness of the ear-piece and the bowl-shaped expansion at the chest orifice; the one advised resembling a funnel. The binaural stethoscope, much used in New York, is also a convenient instrument; it can be adapted, by means of its flexible tube, to any position of the patient, and one of its best claims for use lies in this fact. An objection exists in the fact that it intensifies sounds, so that an untrue estimate may be affixed to them. Training will obviate this objection, and all will do well to secure familiarity with its use.

Normal Vocal Resonance and Vocal Fremitus. When phonation is attempted, the consequent vibrations of air are carried downward as well as upward. The downward path conducts the waves of sound into the fine bronchioles, and thence into the vesicular structure of the lung; thence these sound waves, broken up of necessity into numerous fine vibrations, are transmitted to the chest walls, and can be appreciated by the ear, as vocal resonance. The vocal fremitus is the same vibration demonstrated by the application of the hands to the bared chest, the method being designated as palpation.

To confine the present remarks to auscultation, the vocal resonance in physiological chests bears parallel relation to the analysis of respiratory murmur. In those situations at which vesicular respiratory murmur is located, the vocal resonance is a diffused, low-pitched vibration. In the regions at which the respiratory murmur becomes bronchial, the vocal resonance is termed bronchophony, indicating a concentrated high-pitched

sound: over the trachea the vocal resonance is more concentrated and higher in pitch, tubular vocal resonance. In pathological states the same terms are used, but broncophony or tubular vocal resonance occur in abnormal situations. Intensity or loudness of vocal resonance is not as important as the recognition of the concentrated quality and high pitch—bronchophony—as compared with the diffused quality and low-pitched resonance, which is normal. If vicarious respiration be carried on on one side of the chest, the vocal resonance is exaggerated and is accompanied by bronchovesicular breathing and vesiculo-tympanitic percussion.

Variations. The timbre of the voice modifies the results of vocal resonance and also the base or chest-tones, which are most frequent in males, produce the most sonorous sounds, and, therefore, the most typical vocal resonance and fremitus.

Another precaution relates to the method of phonation. This should be practiced slowly; the numerals, one, two, three, or twenty-one, twenty-two, twenty-three, should be pronounced, so that the vibration developed by each note should be a distinct entity, and each vibration should cease before a second commences.

Whispered Voice. In pathological states of the lung, especially those attended by formation of cavities, it will be found advantageous to study the whispered voice by auscultation, but the vibrations are too feeble to be appreciated in the normal condition of the lung tissue, either by auscultation or palpation.

Palpation. Vocal Fremitus. The practice of vocal fremitus is more simple, since the fingers are incapable of appreciating the many variations of tone and quality which the ear can readily detect. The vibration of the voice can be felt more or less distinctly, as a tremor, which has been compared to the vibration felt over the back of a cat when the animal is purring, and has been termed a purring tremor.

Over those portions of the chest wall where the tissues are thinnest the vibrations are most distinct, naturally in the upper anterior portion of the chest, and the lateral regions, then posteriorly, below the scapulæ. Very marked difference in the amount of vocal fremitus on the respective sides of the chest anteriorly, has been considered pathological; although it has been agreed that the vocal fremitus is usually more distinct over the right apex than over the left. So many conditions influence the conduction of waves of sound in the chest, and since vocal fremitus depends on this conduction, it would appear safest to say that unless the variation is that of marked increase on one or other side, and is combined with other physical signs, the evidence is not to be admitted as testimony of decided change in the textual condition of the lungs. Palpation is also useful to confirm the results of inspection.

Mensuration and Inspection are methods of examination conveniently considered together, since they are usually concerned in the study of the same phenomena; they are the filling out or making more exact the results obtained by the other methods.

The most important result of *mensuration* is found in the satisfactory record which it can yield of the contour of the chest. This is accomplished by taking measurements of the chest at different sectional planes. The tracings can be made by applying to the chest strips of sheet lead, three-quarters of an inch wide and graduated along the border. They can be started from the spine, and end at the median line of the sternum, which point is accurately noted on the strips. It is easy to detach this appliance, by gentle manipulation, without altering its curves. If a large sheet of paper be spread upon a table near at hand the strips can be laid upon it. A pencil is then drawn closely along its curve, a mark made to indicate the position of the spine and sternum, and so we have a precise tracing of the surface of the thorax measured. We can

repeat this for both sides, being careful to place the spinal and sternal points at exactly corresponding points on the first tracing. Besides being a record of contour, this method is an accurate mensuration; and by it we can note the relation between each diameter of the two sides. It is usual to take a tracing at a single level, choosing the lines of the fifth or sixth intercostal space.

If it is desired to record the state of the chest, during full inspiration and strong expiration, to ascertain the extent of the respiratory movements, it will be necessary to make a separate tracing of the chest at different states of respiration.

The graduated tape is a more rapid method, and unless the contour of the chest is to be kept on record, it will be preferable. The tape is to be applied moderately tightly around the chest, from the spine to the mid-sternal line, a mark having been made at the level designed to measure the chest. The number of inches registered by the measurement of the two sides are simply added together and the result read off. If we wish to measure the longitudinal diameter, a line from the clavicle to the base is selected. There may be a slight difference between the two sides of the chest, owing to the habit of the patient in using chiefly the right or left hand; a considerable difference is compatible with the healthy standards.

At full inspiration, the girth of the chest can be increased from two to three inches, a fact of which the method of measurement just described takes cognizance.

Inspection is primarily of value in defining the ideas of size, contour and movements. In size, the normal chest should be symmetrical in each of its parts, as well in shape as in movement. Its characteristics need no detailed description in this place. The difference between the two halves of the chest alluded to in mensuration prevail. Inspection gives a mental picture of departures from the stand-

ard contour which have been classically defined in some peculiarities of shape; notable among these are the phthisical, the rachitic, and the emphysematous chests.

In the *tuberculous thorax*, the vertical diameter of the chest is increased, with marked obliquity of the ribs, and a corresponding shallowness in the antero-posterior diameter. The shallowness is especially marked in the upper portions of the chest, owing to the poor development of the external thoracic muscles, and the consequent imperfect expansion of the upper lobes of the lungs. The infraclavicular regions are deep and pronounced. The clavicles and shoulders prominent and overhanging. Partly in consequence of this, and partly in consequence of the weakness of the trapezius and other muscles of the upper and back part of the chest, the scapulæ are tilted forward, and their angles project markedly. This appearance, when highly developed, well justifies the name given to this alteration of the chest, of "alar," "pterygoid," or winged. A transverse section of such a thorax shows that the elliptical form is greatly intensified, the transverse diameter increased, the antero-posterior are shortened, with a depression corresponding to the external region. This increase in the vertical diameter is intensified by strong action of the diaphragm, which is obliged to contract powerfully, to atone for the insufficient respiratory movements of the upper lobes of the lungs. It is this deficiency, and small capacity of such chests, associated with general constitutional weakness, which predisposes persons with marked alar chests to pulmonary phthisis.

The "flat" chest is often seen independently of the "alar" chest. It may be regarded as indicating a predisposition, or phthisical tendency. It depends upon a change in the shape of the cartilages of the true ribs, which lose their curve, and become straight. The sec-

tional area of such a chest is much changed, and its vital capacity diminished. These alterations of the form of the chest appear to be congenital in many instances, and mark the manner in which, by the transmission of peculiarities of physical conformation, a form of inherited tendency to disease is brought about. In other cases they are acquired, or at least greatly intensified, owing to imperfect expansion of certain portions of the lungs during childhood. The facts are practical, and teach a lesson that care in reference to providing suitable gymnastics for children should be observed.

The Rachitic Chest. Rickets is a disease of infancy and early life, one of the principal features representing a softening of the ribs and their cartilages. The latter represent a less specialized state of connective tissue; manifestly, then, the softest parts of the ribs are near their sterno-costal articulations.

When the diaphragm descends during inspiration, the softened ribs are not able to maintain the distention of the chest until a fresh supply of air enters, at the next inspiration. Consequently, the ribs yield at the costo-sternal articulation, being drawn inward, until a shallow longitudinal groove is formed along the side of the chest, parallel, or a little external to the sternum. Now, if the deformity is uncomplicated by frequent catarrhs, these changes tend to disappear, to a remarkable degree, as the health improves. But the imperfect rarification of the air contained in the lungs promotes pulmonary congestion, and this in its turn predisposes to bronchial catarrh, the sequel of which is the pigeon breast, more or less trace of which can be found whenever the rachitic state is prolonged.

Pigeon Breast. The essential character of the chest is a straightening of the true ribs, which throws the sternum forward, the transverse diameter of the chest becomes narrower, and the appearance is plow-shaped. Rachitis, defective in-

spiration occasioned by chronic catarrhs, enlarged tonsils, etc., have been mentioned as predisposing causes.

The modifications of *emphysema* are noticed in the chapter on that disease.

In studying the *movements* of the chest, there should be both superior and inferior thoracic expansion and retraction, but there should be some diaphragmatic breathing, more marked in the male than in the female. In the latter the superior mobility of the upper ribs is physiological, and obviates the necessity for as much diaphragmatic movement as in the male.

Inspection properly employed becomes a most useful diagnostic method; further deviations from the ideal or individual standard will be indicated in appropriate sections.

Practice inspection with the body in the easiest and most natural position, sitting or standing, with the surface exposed or covered only by a thin, tight-fitting garment.

In inspection, as in all physical examinations, the deviations are not to be compared to an ideal standard theory, but carefully contrast each lateral half with corresponding parts of the chest in the *same individual.* Carefully note in detail subclavicular space with subclavicular space, axillary region with axillary region, etc.

CHAPTER III.

PRINCIPLES OF CLASSIFICATION. CROUPOUS AND ACUTE CATARRHAL PNEUMONIAS.

The object of physical diagnosis applied to the lungs is to ascertain the textural conditions of the pulmonary substance. The analysis of cases by this means may properly be designated the local diagnosis; which, in some instances, may be sufficient to establish the existing pathological process, but in many cases, a study of the etiology and symptomatology must be combined in order to establish a complete diagnosis. The neglect of this method of study has discouraged many investigators in this direction, and has dwarfed the growth and development of this cardinal principle in the investigation and correct appreciation of disease. For purposes of clinical study disease of the pulmonary parenchyma may be grouped in three classes, including both acute and chronic pathological processes, but chiefly the chronic type:—

1. Those cases in which the pathology consists essentially in changes in the vesicular pulmonary structure, including an increased distention of the vesicular wall, sometimes with more or less bronchial inflammation, and, in some instances, with an increase of the inter-alveolar substance. In this class may be placed, as chronic processes, the different forms of catarrhal inflammation of the lungs synonymous with chronic pneumonia or caseous phthisis, catarrhal pneumonia, subacute and chronic, and galloping consumption. In emphysema of the lungs, the vesicular structure is principally involved, although it often has a causative relation with bronchial inflammations. Among the acute forms of pathological process may be designated acute croupous and catarrhal

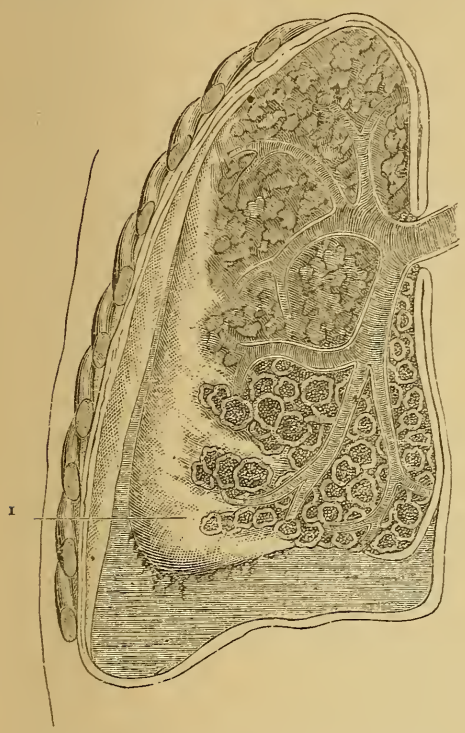
pneumonias, and acute inflammations of the bronchial mucous membrane.

2. Those cases in which the conditions involve special changes in the inter-lobular structure without so much involvement of the vesicular wall, excepting vesicular emphysema, and without essential changes in the bronchial tissues. In this second class belong, primarily, interstitial inflammation of the lungs, designated as fibroid phthisis. In addition, the nodular inflammations characteristic of phthisis are scattered throughout the interlobular structure, causing, by irritation, inter- with peri-lobular thickening, and diminishing the elasticity by increasing the density of the lungs without choking the vesicles, and with or without much bronchial inflammation; synonymous with pulmonary tuberculosis, acute or chronic.

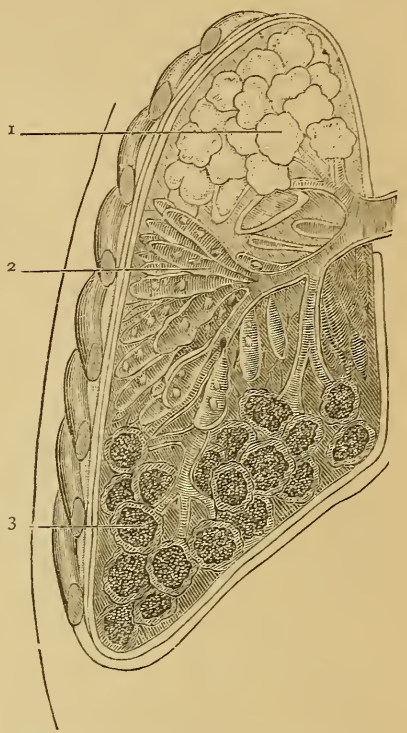
3. Those cases in which the principal pathological alteration occurs in the bronchial tubes, with but moderate structural alterations in the vesicular or inter-vesicular tissue. Under this head belong those forms of inflammation in which the bronchial tissue is specially involved, synonymous with broncho-pneumonia, frequently preceded by repeated colds, and often manifested merely as bronchitis, for years. It is a condition associated with various slight tissue changes, usually in both lungs; but which terminates in that textural loss of substance in one or both lungs which has led to the definition broncho-pneumonic phthisis. Atropic emphysema also belongs in this third group. These conditions have been laid down as occurring separately, but in reality, these forms so interlace, that, practically, they are often difficult to distinguish.

Croupous Pneumonia. The physical signs of this condition are most marked in what is called the second stage, which represents the malady at its full height. In croupous pneumonia there is a strong tendency for the dis-

ease to spread throughout the entire lobe, on this account it is described as lobar, although at first it may only involve a portion of a lobe. In the croupous process the exudation is poured out into the vesicles, which are the only structural parts of the lung primarily involved.* The lower lobes of



Pleurisy, with effusion; condition of pleura; also condensed lung above; patulous bronchial tubes. 1. Croupous or Vesicular pneumonia. The drawing, therefore, represents the process known as pleuro-pneumonia.



1. Supplementally distended vesicles. 2. First stage; crepitant râle. 3. Croupous or Vesicular pneumonia; stage of consolidation.

one or both lungs are usually affected in croupous pneumonia, but it may affect the entire lung upon one side, or any of the lobes separately. Two of the natural conditions of pulmonary substance are violated, elasticity and vesicular air-containing capacity; the bronchial tubes, however, usually

* *See* Catarrhal Pneumonia.

contain air, but the air is confined under an abnormal tension.

Percussion in Croupous Pneumonia. Two changes necessarily result in the percussion note, to wit: its quality becomes very dull, and the pitch very high, since the only air from which resonance can be developed is contained in the bronchial tubes, which are surrounded by dense consolidation. The percussion note is rarely flat, as in pleural effusions, since some air is mostly passing in and out of the bronchial tree. The physical signs by percussion in the initial and terminal stages of acute pneumonia, are that in the outset the resonance gradually increases in pitch and loses in pulmonary quality, until very marked dullness is developed. In the terminal stage the retrogressive metamorphosis of the exudation, leads the type of resonance gradually back to the normal pulmonary.

Deviations. In exceptional cases so much pressure is exercised by the new formation upon the bronchial tubes, that they are compressed, collapse, and little or no air being permitted to enter the affected region, the percussion may become flat.

Sometimes, in pneumonia, especially of the upper lobes, the percussion resonance is neither flat nor very dull; but instead of this, the vesiculo-tympanitic sound, is developed. The cases in which this resonance occurs are those examined early, before the stage of total consolidation is reached. In these cases the first effect of the inflammation is to impair the elasticity of the lungs; its ability to empty itself of air in expiration is crippled, and at each effort at inspiration the distention of the vesicle is somewhat increased; or the sound may be developed from the distended vesicles of a lobe around the focus of inflammation. The sound has been ascribed to the air confined in the bronchial tree in its larger divisions, the resonance being favored by some unusual

distribution or calibre of the tubes; or some unusual thinness of the thoracic walls, rendering vibration of air in the bronchial tubes possible. In most cases the first explanation only is applicable, since the vesiculo-tympanitic sound is transient, and is almost always succeeded by the usual very dull percussion sound. (*See* Respiratory Percussion.)

Auscultation in Croupous Pneumonia. In any acute form of consolidation of the lungs, such as pneumonia, the intensity or volume of the respiratory murmur is increased in contrast with all the chronic forms of consolidation of the lungs, in which the intensity or volume of the respiratory murmur is feeble. The explanation is, that in cases of chronic consolidation, the lung involved is gradually disabled, and slowly but surely the air is diverted to those portions of the pulmonary substance in which it can be most thoroughly aerated, and less and less is distributed to those parts incapable of functional activity. The more important attribute of the respiratory murmur, viz., the quality, now claims our notice. It is readily understood that this must be tubular, since, in the affected district, the bronchial is the only tissue permeable to air. The pitch of the tubular sound is high. The typical tubular breathing is so concentrated as to develop the impression that the air is rushing in and out just beneath the ear. When the consolidation is complete, the echo is brazen or ringing, as though the air were vibrating through metal tubes. The rhythm, the third attribute of the respiratory murmur, must be a come-and-go murmur, of equal length in the inspiratory and the expiratory movements. In the initial stage of pneumonia the increment of bronchial breathing up to the tubular sound is gradual, and in the terminal stage its retrocession to normal vesicular respiratory murmur is slow.

Râles in Croupous Pneumonia. The dry, crepitant râle is heard during the initial stage of the process, but invariably disappears when consolidation is complete. One must, there-

fore, auscult very early in the case, to secure this râle, but sometimes the area of pneumonia extends, and then crepitant râles precede the involvement of new districts of tissue. The stage of resolution is characterized by a sub-crepitant râle, due to the softening of the exudation.

Deviations. The first deviation relates to rhythm. The breathing is none the less tubular or intensely bronchial, although only inspiration can be detected. Hence the importance of appreciating the quality and pitch of respiration as fundamental. It is often the case that when pneumonias pass into the third stage, and resolution is delayed, one can hear only the inspiratory movement, and the longer the consolidation persists the more likely is this to be the case. The second deviation relates to intensity, and is subject to the same rule; the longer the consolidation persists, the less intense is the respiratory murmur, since, naturally, the air is more abundantly distributed to those portions of the tissue best adapted to oxygenate it.

Vocal Resonance in Croupous Pneumonia. Having ascertained the quality of the respiratory murmur, the vocal resonance is readily classified. The sound is termed bronchophony, but it may be so concentrated in quality and so high pitched as to be termed tubular bronchophony. If the timbre of the voice is very high, or the bronchial tubes are compressed by exudation, transmission of the waves of vibration after phonation is not appreciable.

Vocal Fremitus in Croupous Pneumonia. The fremitus is increased in all typical cases; viz., those in which consolidation is equally distributed throughout a section of lung. If the timbre of the voice be high and the chest walls thick, the fremitus is correspondingly feeble. If the chest walls are thin, the fremitus is more perceptible. Sometimes, owing to the large amount of air entering the unaffected portions of the lung, the volume of vibration is distributed in such ample

waves to these healthy portions, as to render the results of vocal fremitus negative. The regions over which the vibrations are distributed with maximum intensity in the normal state of the lungs must be kept in mind while making these observations.

Summary. We have as typical signs of the complete consolidation of a district of lung tissue, very marked dullness on percussion, tubular breathing, tubular bronchophony, and increased vocal fremitus. In children who possess very thin chest walls these physical signs are even more marked. For differential diagnosis between croupous pneumonia and pleurisy, see section on pleurisy.*

Terminations of Croupous Pneumonia. In croupous pneumonia resolution is usual in from five to ten days. The exudation disappears, partly by absorption, partly by expectoration, through the medium of a rapid fatty degeneration. If, however, this favorable change does not happen, the resolution may be delayed, and yet occur after the lapse of two or three months, meanwhile the lung remaining in a state of gray hepatization. Frequently, however, the gray hepatization passes into a state of purulent infiltration. The tongue becomes more or less dry, fever of hectic type sets in, recovery may occur, but death from exhaustion is more common. Exceptionally, the formation of excavations of the lung complicates this stage, and the process becomes an unusual form of phthisis, to be diagnosticated chiefly by the history.

There are two symptoms which belong essentially to the initial stage of croupous pneumonia; one is the disappearance of the chlorides simultaneously with the advent of the process, and the other is hæmoptysis, which is not an essen-

* The differential diagnosis includes pleurisy with effusion, pleuropneumonia, and the forms of catarrhal pneumonia. See appropriate sections.

tial phenomenon, but is frequently a symptom when pneumonia sets in among the young or vigorous

Acute Catarrhal Pneumonia.* In croupous pneumonia the radicles of the pulmonary arteries are chiefly the sources of the inflammatory exudation into the vesicles; in catarrhal pneumonia the radicles of the bronchial arteries are chiefly concerned. The disease consists in the intertwining of descending bronchial catarrh with thickening of the vesicular wall and inter-vesicular substance, but with only a partial occlusion of the vesicle with cellular proliferation, composed of leucocytes, corpuscles from the inter-vesicular connective tissue, together with epithelial proliferation. It is often associated with areas of atelectasis. The vesicles not being absolutely occluded, remain somewhat pervious to air, and the process itself is diffused over the lungs, although there is a special tendency to involve the apices. Contrasted with croupous pneumonia, which always involves more or less completely an entire lobe, we find that catarrhal pneumonia is usually distributed in the upper lobes in one or both sides of the chest, but it may involve areas or patches distributed through the middle or lower lobes. This characteristic may be explained by the etiology of catarrhal pneumonia, viz: a descending bronchitis. There is not the same degree of solidification as in croupous pneumonia, unless it be over a very small area.

Percussion in Acute Catarrhal Pneumonia. The elasticity of the lung is seriously modified, the tension of the contained air is increased, and the result is elevation of percussion pitch, with usually a dull quality, proportionate to the extent of the invading process, but the percussion is never flat. The quality of the resonance may be rendered slightly tympanitic by areas of vesicular emphysema interspersed among areas of consolidation.

Auscultation in Acute Catarrhal Pneumonia. In proportion

* For chronic forms of Catarrhal Pneumonia see Phthisis.

to the juxtaposition of the areas of consolidation the respiratory murmur is bronchial or broncho-vesicular. The volume or intensity depend upon the amount of air by which the diseased area is permeated during inspiration or expiration, and upon the completeness of the respiratory acts. The respiratory murmur is in marked contrast with the tubular brazen sound heard in the fully developed croupous process.

Râles in Acute Catarrhal Pneumonia. Fine subcrepitant or small mucous râles will usually be heard over a widespread area during the course of the disease, as the evidence of associated capillary bronchitis.

Vocal Fremitus and Resonance, in Acute Catarrhal Pneumonia. These conform to the rule that the more dense the consolidation, the more pronounced the vocal resonance and fremitus.

Terminations in Acute Catarrhal Pneumonia. Catarrhal pneumonia pursues a slower course than croupous pneumonia, the physical signs and febrile phenomena disappearing gradually as the resolution progresses; or, the process may advance until the tissues become more and more choked with cells, by which the radicles of the bronchial arteries are compressed, the circulation interfered with, necrosis induced, and, in adults, a slow or rapid phthisis commonly results. Acute catarrhal pneumonia, followed by resolution, is frequent in children, and is a possible though improbable termination in adults. At any age there is a tendency to recurrence of bronchitis.*

In acute pneumonias, whenever the aerating surfaces are curtailed, dyspnœa is a marked symptom, but in subacute or chronic processes the supplemental action of the uninjured pulmonary tissue, or the diminished demand made by the system for oxygenated blood, owing to a reduction of the systemic vital activity, result in removing this symptom

* *See* Capillary Bronchitis and Desquamative Pneumonia.

from consideration. If dyspnœa remain a symptom it is connected with exertion upon the part of the patient. Again, in all acute pneumonic pulmonary processes, a dark red flush may often be seen upon one of the cheeks, frequently, but not invariably, on the affected side.

We next pass on to the consideration of the subacute and chronic consolidations alluded to in the classification at the beginning of this chapter, leaving the various forms of bronchitis and emphysema to be described subsequently.

CHAPTER IV.

THE MORPHOLOGY OF PULMONARY PHTHISIS. SYPHILIS OF THE LUNG.

Next to the question of etiology, the morphology of pulmonary phthisis is most interesting to the diagnostician, because a comprehension of pathological processes is fundamental to the correct interpretation of physical signs. In certain individuals, owing to inherited tendency or particularly unfavorable surroundings, recovery after bronchitis due to cold is retarded, or a susceptibility to a new bronchitis is increased. In either case, lingering catarrh, in the majority of instances, is the inception of the series of pathological processes known as phthisis pulmonalis. Two macroscopical appearances, nearly identical, occur; but they differ materially in their microscopic anatomy. Certain so-called miliary tubercles are frequently composed only of the inflammatory products of connective tissue, without the characteristic true tubercular arrangement. The word miliary expresses their nodular appearance, but the inflammatory products may be so arranged as to represent true tubercle, pseudo-tubercle, or a diffuse inflammation. This tubercle, or pseudo-tubercle, is constant in the different forms of phthisis in the adult, except in the instance of pure interstitial pneumonia. Frequently the amount of tubercle tissue in the lungs is so great as to form the principal part of the process, although complicating inflammatory cheesy products are also present.

Tubercular peri-bronchitis is probably the best term for the earliest stage of phthisis, and sometimes is the best to designate the process all the way through. The appearance of the peribronchial tissues resembles berries on a stalk. The formation extends along the bronchi, spreading from acinus to acinus

until the trunk is reached, and is also distributed in the sheaths of vessels and lymphatics. True tubercle may penetrate a bronchus and involve the lining membrane, and a true tuberculous ulcer may form the basis of an extensive associated bronchitis. Aside from this, these infiltrations excite interlobular connective tissue growth, and subsequently, the walls of the vesicles become thickened, and some vesicular catarrh ensues, which may occlude a lobule. Thus the three divisions of the pulmonary tissue share in the pathological process of primary phthisis

Desquamative Pneumonia. The cause of vesicular consolidation is usually a process of desquamative pneumonia. This term is used to describe the diffuse inflammation which may accompany the former processes, and which, more than the other changes, paves the way for the disintegration of the lung. In this process the peripheral epithelial cells of the bronchi are proliferated and shed, thus filling the bronchi and infundibula, while the surrounding connective tissue becomes infiltrated with cells. In children this process is a common one, on account of the greater cellular activity in these subjects. In the desquamative catarrhal pneumonias of early life the process differs from the desquamative pneumonias of adults in that the air-cells only are filled, and there is little or no change in the intervesicular tissue. Hence it is that recovery is so much more frequent in children than in adults. This process of desquamative catarrh is the basis of those cases of acutely developed phthisis which follow croupous or catarrhal pneumonia, and which has been called acute catarrhal phthisis, or galloping consumption—sometimes pneumonic phthisis. When the pathological process thus described is less rapid, the result which follows has been classed by some as acute caseous pneumonia. When the changes are still more slowly developed, it is synonymous with the chronic catarrhal pneumonia. The relative development then of these processes,

known as phthisis, in the three divisions of the pulmonary tissues, the bronchial, the inter-lobular, and the vesicular, is dominated by the activity of the inflammatory process; peribronchitis, with consecutive changes in the latent forms, desquamative pneumonia in the more acute forms; while a process presently to be alluded to—interstitial phthisis—occurs as a very slowly developed change. In many cases when the destruction of the lung is very rapid, the tubercular deposition, true or pseudo, occurs in the early stages but is masked in the later by the development of the secondary inflammatory desquamative pneumonic processes.

The situation at which phthisis is developed is probably most frequently the apex. The forces of expiration presumably are less efficient at this site, and the lungs are less entirely freed from mucous. The circulation also is less than at the roots, and the products of inflammation are therefore dryer. The roots of the lungs, however, in a large minority of cases are primarily affected. This is especially true of cases in which the original processes of invasion are latent. It is a clinical point of some interest, that when the area of the lung involved in the process of phthisis is distinctly limited, and does not shade off gradually into healthy lung a favorable result may be possible. Certainly, the process of phthisis may be arrested, even when it has passed into the stage of cavity, if the lesions are sharply defined. The localization of a lesion, other things being equal, is a point of favorable prognosis. Another topic of interest is the causative share taken by pleurisy in the development of phthisis. But, while this is true, it is conceded that dry pleurisy is a frequent secondary lesion in the progress of pulmonary tuberculosis. Pleurisy is, in many cases, very conservative, since, by the thickening and adhesion of the pleural surfaces, the ulceration of the walls of superficial cavities is arrested and pneumothorax is prevented.

Interstitial Phthisis, or *Cirrhosis*, is a process in which true or pseudo-tubercle may or may not be associated. The pulmonary tissues are traversed by narrow bands of connective tissue, which may gradually compress it more and more, finally converting it into dense fibrous masses. The color of the lung is apt to be slaty and dark. However, in some cases of interstitial phthisis, patches of the peri-bronchial and desquamative pneumonia may be found, with cheesy degeneration. Interstitial formation is an important part of the process by which cavities are enclosed and sometimes cicatrized. Interstitial phthisis, if consecutive to bronchitis, is usually bilateral, affecting the upper lobes, although as a unilateral affection, it is not infrequent.

Acute Miliary Tuberculosis. A synopsis of the pathology of phthisis would be incomplete without including some mention of acute miliary tuberculosis. This process may be primary in the lungs, or secondary, as a part of the general infiltration of the serous and mucous membranes, the lymphatic glands, and the viscera. This form of tubercle is characteristically recognizable only in the miliary stage, as a small, hard, translucent nodule.

Pathology of Syphilitic Phthisis. The early lesions of so-called syphilitic phthisis are evoked very often by bronchial catarrhal inflammation, which predisposes to structural changes. In these cases there is a principal interstitial fibro-nuclear growth commencing in the alveolar wall and concentrically arranged around the smallest bronchi and pulmonary vessels. Wagner maintains that the alveolar wall is implicated as commonly in syphilis as in ordinary phthisis. Narrowing of the bronchial tubes occurs in syphilis of the lung by the pressure of the new growth which develops along their lumen; occlusion may occur from this new formation, but it is also caused by the enlarged bronchial glands, one of the effects of syphilis. By this means, serious mischief

in the lungs may be developed, in kind proportioned to the degree of obstruction, such as atelectasis, emphysema, and certain forms of pneumonia. Further, in the effect of interstitial processes upon the bronchial tubes the syphilitic differs from the non-specific disease. The tough, contracting, fibrous tissue which radiates through the lung draws together the bronchial tubes, and deforms by narrowing or flattening them, possibly even to obliteration. On the contrary, in the forms of non-syphilitic fibroid phthisis the bronchial tubes are widened. The process proceeds, in syphilis, from the hilus into the interior of the lung, following the distribution of the bronchial radicals and the bronchial arteries. The lesions also occur on the surface near the visceral pleura, where there is also more connective tissue. Green and Virchow suggest that the origin of syphilitic diseases of the lung is distinctive in this respect, that while in ordinary phthisis the fibroid is secondary, or co-equal in its development with changes in the alveoli and alveolar wall; in syphilis there are primarily interstitial changes. Later, when entire vesicular consolidation and breaking down occurs, the process is similar to ordinary phthisis and indistinguishable from it. The vascularity of the new growth of connective tissue is also claimed to be a distinctive characteristic of the inflammatory proliferation due to syphilis. But the one of the characteristic and special lesions of syphilis is the change in the intima of the blood vessels. This has not yet been demonstrated in the lung, but merely general thickening of the external coat of the vessels.

Gummata may be produced anywhere in the intervesicular tissue, usually near the visceral pleura. They occur in the deeper layers of the costal pleura or on the periosteum of the ribs. Owing to their peculiar anatomical formation their subsequent history is one of combined caseous and fatty degeneration. Fibroid development may ensure their ad-

hesion to the visceral and costal pleura, and they may point externally, with or without many of the appearances of inflammation in adjacent tissues; or they may open internally, resulting in the formation of cavities; or they may remain stationary for an indefinite period.

Enlargement of the Bronchial Glands is another important incident of the syphilitic process, and is observed to a moderate degree in the ordinary phthisical process.

Symptoms of Syphilitic Phthisis. The symptoms are most insidious and gradual in their onset, and may be classified as the subjective, the physical signs, and the objective phenomena. The subjective symptoms are possibly evidences of mediastinal pressure, without noticeable departure from an appearance of health. There may be difficult respiration, with more or less dyspnœa, especially in mornings and evenings, besides a sense of heaviness and oppression in the chest. These symptoms are increased by exertion; respiration may become wheezing, with imperfectly developed asthmatic attacks. Hoarseness, aphonia, dysphagia, and unequal pupils may be present. These symptoms may be interlaced with the cachexia produced by the early and later stages of systemic infection.

Syphilitic disease of the larynx may occur co-equal with the pulmonary trouble, and some of the above symptoms may thus be explained. If there be much enlargement of the bronchial glands there is a sense of discomfort, oppression and uneasiness at the root of the neck, which increases until actual pain is felt located in the back between the scapulæ, sometimes radiating through the intercostal nerves around the chest. Cough, as a rule, is an early symptom, usually dry, paroxysmal, and interlaced with dyspnœa, but there may be bronchial catarrh with a relative amount of expectoration. When a physical examination of the chest is instituted, thickening of the head of the periosteum of one or both clavicles,

substernal tenderness, thickening of the tibial periosteum, are usually detected. The pulmonary lesions may develop primarily, but frequently are associated with enlargements of the bronchial glands.* The physical signs in syphilitic phthisis unassociated with gumma are, on auscultation and percussion, those shared by other forms of fibroid phthisis—increasing dullness, varying degrees of bronchial breathing and bronchophony. When cavities occur the physical signs necessarily correspond to those of other varieties of phthisis in this stage. A peculiar alveolar rustle resembling the sound produced by the rumpling of wall paper has been described. By inspection some of the changes similar to those in fibroid phthisis may be found, also displacements of the heart, or trachea. When a gumma is large enough to be recognized by physical examination there is absent vocal fremitus, flatness on percussion confined to a section of the chest and not occupying its semicircumference, as is usual in pleural effusions. The respiratory murmur is abruptly cut off over the area of flatness, but there may be distant bronchial breathing. The vocal resonance is absent, or distant bronchophony. Around the gumma the respiratory murmur is usually very feeble or scarcely audible, râles may be present, on account of neighboring congestion. Proportionate vicarious functional activity prevails in the opposite lung. If the gumma be large the heart's impulse may be displaced to the left or right, and dyspnœa occur, as it would in pleural effusions. In this stage, owing to irritation of the bronchial mucous membrane, there may be expectoration of a tough, glairy mucous, or as the gumma softens the expectoration may become purulent. Among the objective phenomena is hæmoptysis, which occurs infrequently, because the process in the lungs is chiefly fibroid, but when cavities form hæmoptysis can occur, as in other forms of phthisis. Bronchial pressure may cause vesicular collapse, with repletion of

* For physical signs of enlargement of the bronchial glands see chap. xiv.

the pulmonary vessels, together with a form of pneumonia which has been described by Fuchs as apneumatosis. Hæmoptysis is possible through the rupture of newly developed blood vessels in the new formation in the lung. The thermometer rises but little, if any, above the normal standard. In physique the ordinary characteristics of a phthisical subject may be wanting. The chest is often well developed and the ensemble is rather that found in fibroid phthisis or pneumonokoniosis. Diarrhœa and night sweats are said to be less frequent in the early or middle stages than in ordinary phthisis, and the pulse is less frequent. The patient may be capable of hard physical labor, but in some cases the complexion is pallid, waxy, indicative of cachexia associated with digestive disorders, there are also night sweats with variable but low thermometrical record. In these cases the systemic poisoning is grave, and many other organs are apt to be coincidently involved, but the process peculiar to syphilis is long past and the patient suffers from simple catarrhal phthisis with formation of cavities or softening gummata. The diagnosis depends mainly on the history, the prior or co-existing syphilitic systemic lesions, especially laryngeal, cutaneous syphilides, exostoses, substernal tenderness, thickening of the periosteum of one or both clavicles. Family immunity from phthisical tendency, recovery from lesions usually incurable if they have other origin than syphilis, are suggestive. Distribution in the middle and lower lobes was found by Grandidier in twenty-seven out of thirty cases believed by him to be specific phthisis. The surrounding lung contained large areas free from disease. By some excellent authorities the existence of pulmonary syphilis is questioned as distinguishable from phthisis induced by the deterioration of the general health as a part of the syphilitic process.

CHAPTER V.

DIAGNOSIS OF SUBACUTE AND CHRONIC CONSOLIDATION.

Early Diagnosis of Phthisis—*Percussion.* The tissue alterations in phthisis, so soon as they can be recognized with certainty, include changes of resonance noticeable in the pitch and quality. In the involved area the pitch of the percussion note is elevated as compared with other regions, especially with the corresponding half of the chest.. The quality of the resonance verges more and more into the vesiculo-tympanitic type. Both these departures from the normal quality and pitch are dependent on diminished elasticity and increased tension in the pulmonary tissue in the involved district. Perfect expiration is largely dependent on the inherent resiliency of the lungs, and the loss of this function implies that in both stages of respiration more air is confined in the diseased pulmonary district than is normal.

Auscultation in Incipient Phthisis. The variation from the standard is mainly in the direction of rhythm, but includes a change in the quality. The study of rhythm usually reveals prolonged expiratory movement; the change in quality soon follows. The statement of two facts explains the conditions. First, pulmonary elasticity is diminished, vesicular collapse is, therefore, slower; and second, the bronchial portion of the expiratory act is much obscured by the slow vesicular collapse; consequently we note the result as prolonged expiration. Sometimes the rhythm is termed interrupted or cog-wheeled, because the modification of the elasticity of the lung prolongs the inspiratory and expiratory period so much that the breathing appears interrupted, jerking, or cog-wheeled. More inspiratory force is required than pertains to a single effort at inspiration, and therefore, more than one effort must

be made before complete inflation of the lungs occurs; similarly the expiration is accomplished in a spasmodic manner. Prolonged expiration may also be due to a loss of pulmonary elasticity in cases of supplemental breathing, or in emphysema; the distinction between this species of prolonged expiration and that due to consolidation depends in the latter case on the high pitch of the expiratory sound.*

The modification in quality consists in the murmur becoming increasingly concentrated and harsh. The pitch of both movements, but especially expiration, becomes higher than normal. This can be understood when we comprehend that the more dense the pulmonary tissues become, the more easily they transmit the sound occasioned by the transit of air in the bronchial tissue. We lay stress on this point, since the change in pitch is a cardinal feature of prolonged expiration associated with consolidation.† A valuable evidence in the early diagnosis of phthisis which is sometimes present is a murmur in the subclavian artery, occasioned by the pressure from the solidification of the lung upon the artery.

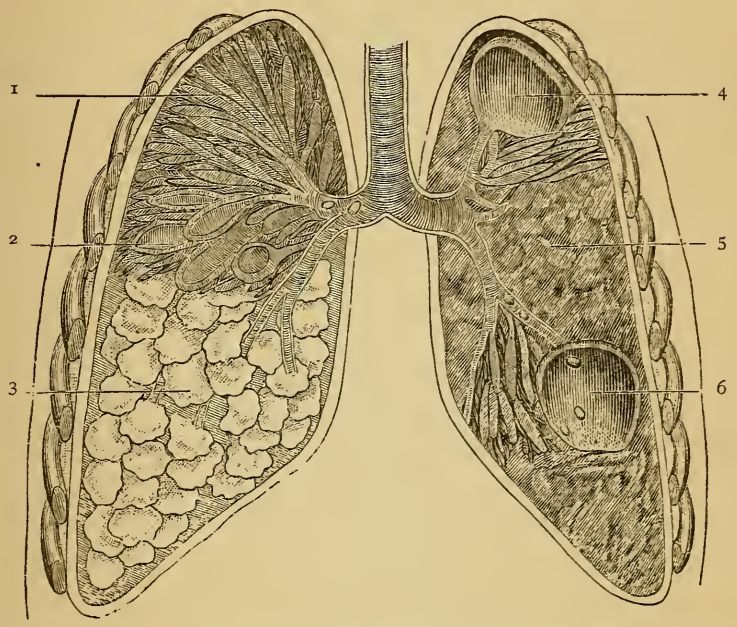
Vocal Resonance and Fremitus in Incipient Phthisis. The quality of vocal resonance is more concentrated than normal, and higher in pitch. The vocal fremitus is often more distinctly felt than is physiological. But in incipient phthisis the lesion is so masked by nearly normal lung tissue, that these signs may not be positive, and sometimes the other regions of the chest containing supplementally acting lung may

* Bearing in mind that auscultation is a study based on the comparative examination of the two sides of the chest, in each individual case; since the quality and pitch of each person's respiratory murmur is a law unto itself.

† The presence of fine crackling or other moist râles merely indicates associated bronchial catarrh, which is present in these cases, more or less, early in their history. Their localization and persistence in connection with the above physical signs, is strong corroborative evidence of incipient phthisis. (*See* chapter on râles.)

yield results which render these methods of investigation negative.

Inspection in Incipient Phthisis. As compared with the normal chest the expansion may be diminished, since the condensation of the pulmonary tissue diminishes its power to resist the extra-thoracic atmospheric pressure; but yet in some cases the tissue adjoining the area of consolidation may



1. Chronic Phthisis—inter-vesicular changes. Vesicular thickening, and collapse. 2. Vesicular Emphysema, in patches overlying areas of consolidation, or intermixed with such areas. 3. Vicariously acting lung. 4. Formation of cavity. 5. Consolidation. 6. Large cavity—amphoric râles or jingles; also in bronchial tubes. See Râles.

become vicariously emphysematous, and so maintain the contour of the thoracic walls, or even cause moderate prominence of the diseased side. It is well to view inspection as of importance to the diagnostician; but slight deviations from standards of comparison must be supported by testimony drawn from other methods of investigation. In using inspection as a method of diagnosis, always fix the eyes on the same relative

points on the opposite sides of the chest, for a comparison of the respiratory movements of expansion and retraction. The clavicle, or line of the ribs are the best landmarks.

Mensuration. It is obvious that this method can only be of real service after phthisis has advanced to a material extent.

Advancing Consolidation. There are three serious departures from the healthy standard in the analysis of a case in which diffused consolidation exists as an advance on the prior condition: (*a*) The lung is more or less increasingly incapable of containing air. (*b*) The confined air is maintained in an increased state of tension. (*c*) The elasticity of the pulmonary tissue is more and more seriously impaired.

Percussion in Advancing Consolidation. The resonance is always abnormally high-pitched in these cases, since the decreased amount of air and the changed physical condition of the tissues combine to produce this result. The quality is subject to an interesting variation; it may become sub-tympanitic, or dull, according to the exact condition of the tissues. If the area over which percussion is practiced is one of consolidation, the quality will be dull. If there be much vesicular emphysema, or if the vesicular walls are largely infiltrated, and yet the vesicles are capable of being over distended with air, then the resonance may be sub-tympanitic. By viewing the question of resonance from this standpoint, the statement can be advanced, that in proportion to the amount of consolidation the percussion will be dull, very dull, almost flat, and the pitch will be high; or the dull note will be replaced over more or less extensive areas by a tympanitic or sub-tympanitic note.

Sources of Error. Care must be taken to avoid being misled by small patches of vesicular emphysema overlying or surrounding areas of consolidation. The frequent location of vesicular emphysema is over the anterior surface of the

upper lobes, effectually masking the condition beneath unless a careful examination is practiced. The examination of the left chest by percussion is complicated, in some cases, by the distention of the stomach to an unusual size. It can encroach on the thorax as high as the third rib in the axillary region. The tympanitic resonance is sometimes so low pitched as to render percussion a negative sign. The distention of the stomach can be detected with the aid of auscultation, by causing the patient to drink a small quantity of water, which, when it drops into the stomach, will occasion a tinkle, very easily detected by auscultation in the left axilla. Another source of confusion is found in those individuals in whom, from any cause, the tissue forming the chest walls becomes thin and tensely stretched over the chest; a careful method of percussion is the best resource under the circumstances. Practice light percussion, so that delicate modifications of pitch may be noted. Direct percussion upon the clavicle without a pleximeter will often be a helpful method; one can make very accurate deductions as to the condition of the entire side from percussion made on this bone.*

Percussion of Comparatively Normal Areas. In the pulmonary substance areas of vesicular emphysema may be scattered through areas of consolidation. The frequent location of this lesion is the anterior surface of the upper lobe. According to Louis, most commonly the right apex or the

* In general terms bone tissue yields a dull note on percussion. If the bone is flat, or nearly so, and encloses a considerable amount of air-containing tissue, *i. e.*, the ribs, the resonance is high pitched and the quality is sub-tympanitic rather than dull, as compared with the remainder of the lung. This happens because the pulses of air are shorter, since the shock of percussion is diminished by the bone. If the resonance over bone acquires a very dull or flat quality, there is probably more or less consolidation beneath. These points pertain to such bones as the ribs, clavicle and sternum. The scapulæ are largely enveloped by muscle, so that the resonance is always dull.

left base is involved. The tympanitic resonance in this condition of lung may be so distinct as to generate the suspicion of formation of cavity. Auscultation must be invoked to decide the question. The difference between hollow breathing and the exaggerated or broncho-vesicular is beyond dispute. To further illustrate the point of supplemental distention of the lung, when a single lobe or more upon one side is functionally disabled, the balance of the tissues substitute in carrying on respiration. The more acutely the consolidation is developed, the more markedly will the substitution be evidenced to percussion. The modifications in the tissues brought about by this vicarious action depend entirely upon the length of time the substitution is required to continue. In acute pneumonia the opposite uninvolved pulmonary tissue is merely hyper-distended and returns to its normal textural state with convalescence from the disease: the same is true when there is a pleural effusion filling one of the pleural cavities, the uninvolved lung acts supplementally. But in chronic conditions of phthisis, or pleural lesion, atrophic intra-lobular changes result more or less completely, in the development of substantive emphysema.

The percussion resonance is the vesiculo-tympanitic, more or less high-pitched according to the loss of elasticity, and the increase of intra-pulmonary tension.

Auscultation in Advancing Consolidation. In harmonizing the results of auscultation with percussion, we observe that in proportion to the amount of consolidation present, the respiratory murmur advances from the harsh or broncho-vesicular to the purely bronchial. In a typical case of diffused vesicular consolidation, it is the concentration of the respiratory murmur together with the elevation of the pitch, which enables us to discriminate. Fancy vesicular consolidation so closely distributed throughout an area of lung that the respiratory murmur is restricted to the bronchial tissue; necessarily

it is very concentrated, very high-pitched, nearly tubular. But in catarrhal phthisis the solidified vesicles are scattered through the diseased district, areas of vesicular emphysema abound, and so the respiratory murmur is rarely tubular, but is mostly classed as some kind of bronchial breathing, according to its degree of concentration and elevation of pitch.

In cases of broncho-pneumonia and fibroid phthisis the respiratory murmur is bronchial, because there is an undue proportion of non-elastic tissue, causing induration of the bronchial and intervesicular tissues. These forms of phthisis are often associated with more or less vesicular emphysema. On this account the respiratory murmur is modified, and sometimes may be called broncho-vesicular.

Rhythm in Advancing Consolidation. The inherent resiliency of the lung tissue is essential to a physiological rhythmical performance of respiration. The proper expansion of the lobes depends very much upon the descent of the diaphragm, and the expansion of the base of the chest. The longer the lesion persists, especially if it be unilateral, the more complete is the supplemental action of the unaffected side, and thus diaphragmatic breathing is not as much exaggerated as inferior and superior costal respiration. Further, the pulmonary circulation depends largely on aeration of the blood; the circulation will be most active at those sites at which the best aeration can be effected. Consequently, when extensive diffused areas of consolidation exist, the respiratory murmur will be most marked in the areas where there is least change. We often hear only the inspiratory portion of respiration, or respiration may be heard on inspiration or expiration, but is superficial. This is on account of deficient diaphragmatic movement and a lessened demand in chronic phthisis upon the respiratory function.

If but a part of the respiratory murmur only is audible, yet, if the quality is concentrated, and the pitch high, the

breathing can be called bronchial. It is especially in the posterior portions of the lungs that the respiration is lost during the expiratory movement. In cases of atrophic emphysema interlaced with phthisis, the respiration is especially feeble. To describe this modification of the respiratory murmur we would say that feeble respiration implies a want of volume or intensity in the complete respiratory act. To resolve doubts as to the pathology of these cases of feeble breathing, command the patient to cough or to take a series of quick inspirations and expirations, which will enable the observer to adjudicate the pitch and quality of the respiration.*

Auscultation of Comparatively Normal Areas. The supplemental respiratory murmur is that type known as exaggerrated, or broncho-vesicular. It is best appreciated by contrasting the idea of elasticity of the pulmonary substance, derived from the examination of normal areas, or areas of vicariously acting lung, with the changed elasticity and increased density of truly pathological pulmonary tissue. The murmur may be very loud, and yet no textural alteration is indicated, but simply an overplus of air and consequently slight increase of tension and slight loss of pulmonary elasticity. The diagnosis often requires the correlation of evidence from all the means of physical diagnosis.

Local Emphysema. Usually the upper lobe, especially the right, or the inferior lobe (Louis), undergoes this alteration. When there is an area of consolidation beneath the emphysematous portion, bronchial breathing is a deeply seated sound, or is best heard by examining the same lung on the opposite side.

* Summary of Causes of Feeble Breathing. Obstruction to the entrance of air into the bronchial tubes, extensive pathological deposits in the pulmonary substance, feeble breathing, associated with pleural adhesions. General substantive emphysema.

Vocal Resonance and Fremitus in Local Emphysema are either negative or can be studied on general principles.

Advancing Consolidation (*continued*)—*Vocal Resonance and Fremitus.* Increased tension and loss of elasticity combine to favor the increased conduction of vocal resonance and fremitus; the more pronounced these departures from the standard condition of healthy lung, the more definite do the results of vocal resonance and fremitus become.

Vocal Resonance. It has been implied that in advancing consolidation vocal resonance is heard more or less distinctly as bronchophony, but confusion sometimes arises, from the following facts. Where lesions are scattered, the intensity of vocal resonance may seem equal on both sides. It is chiefly differentiated by giving attention to the concentration of the sound, which, with the elevation of pitch and bronchial quality, are the peculiarity of bronchophony. Exaggerated vocal resonance is merely intensely loud vocal resonance of normal pitch and quality. This bears out the remark that intensity is an attribute of vocal resonance which is of subordinate importance. The recognition of vocal resonance sometimes extends the key to the proper appreciation of the variety of respiratory murmur present, and should be practiced if doubt exists as to the quality of breathing.

Vocal Fremitus. The best situations for the recognition of vocal fremitus are the locations at the apices and the axillary regions. The exceptions to the usefulness of the sign for diagnostic purposes are to be found in the fact that in many cases of condensation of the lungs the process, if advanced, so far precludes the admission of air into the lungs as to prevent the study of vocal fremitus. In addition to this, if there be much disseminated emphysema, or if there be any broncho-pneumonia or fibroid phthisis, the evidence from palpation is apt to be negative.

Inspection in Advancing Consolidation. Its evidences are

dependent on the physiological facts that the distention of the chest is maintained by three factors: the existence of the air within the chest, the elasticity of the lungs, and the inherent resiliency of the thoracic walls, all resisting atmospheric pressure from without. In phthisis the two first factors are withdrawn; the result reveals a deficient expansion with inspiration, and a retraction proportionate to the amount of condensation of the lung substance and dependent on the want of resistance within the chest to atmospheric pressure from without. The retraction is most apparent at the apices, since lesion is chiefly developed in them, but in addition, at the inferior portions of the chest retraction sometimes occurs, from consolidation, but more frequently from pleurisy.

Inspection is also helpful in enabling us, sometimes tentatively, sometimes positively, to differentiate between classes of phthisis. The greater the amount of fibroid tissue developed in the type of phthisis, the greater the amount of external alteration of contour will result. Not only so, but displacements of the intra-thoracic movable organs will occur. In fibroid phthisis we have an exception to the rule assigning retractions of the base of the chest to pleurisy; the contractile power of the newly developed cicatricial tissue is so great that thoracic resilience cannot contend against it, and retractions occur. Broncho-pneumonia develops a tendency to those changes in the appearance of the thoracic walls typical of emphysema, elsewhere described, but without equal distention. Fibroid phthisis presents retractions already indicated, but also displacements of the heart toward the affected side, which may be so pronounced that the apex beat will be carried into one of the axillary spaces, and upward as high as the second interspace in that region. The trachea may be displaced to the one or other side, and the dip toward the abnormal side can be both felt, as well as noticed. It often happens that the processes of fibroid

phthisis and broncho-pneumonia are blended. In this case, the evidence by inspection partakes of both types. The importance of the clinical study of the associated symptomatology and etiology in the diagnosis of these types of disease is very great.

Finally, inspection leads us to notice the type of chest presented. The phthisical chest has been represented as long, flat, coffin-shaped. The shoulders stoop, the spine is carried forward, the ribs are more oblique, depressing the sternum, elevating and tilting outward the scapulæ, the result giving the alar or winged appearance. There is this much to be said relative to the share of this contour of the chest in the development of phthisis. The chest capacity is reduced by the awkward position of the muscles, and when catarrhs develop they are more apt to become chronic. Beyond this, the importance of this shaped chest as a predisposing factor to phthisis has been exaggerated.

Exceptional Phenomena in Advancing Phthisis. When there is pressure on the bronchial or laryngeal tissue, the expansion of the lung does not follow the expansion of the chest walls, which are carried outward by their inherent elasticity, so that the intercostal tissues are depressed. There is another condition under which the intercostal tissues move inward on inspiration, viz., in many cases of pleurisy with and without effusion, as will be explained in the section on Pleural Alterations. This is quite apart from the condition of retraction in chronic pleurisies with adhesions, in which retraction of the interspaces occurs notably with the expiration. (See Inspection, in chapter on Emphysema.)

Mensuration in Advancing Consolidation is in most respects the complement of inspection; the value of tracing a plan of the chest on paper is to make a record for reference. Circumferential measurements are not to be relied on, save in cases of extreme retraction; for further comments on this subject

consult section on unilateral enlargement, under title of Emphysema.

Hypostatic Congestion. The grade of bronchial breathing is appropriate to the physical conditions (amount of condensation) present. But the diagnosis is an example of the associated study of clinical environment. Hypostatic congestion supervenes during the typhoid state in fevers, also in those long bed-ridden, with feeble hearts. Sometimes it occurs in the course of acute rheumatism. These points, with the bilateral distribution, separate it from acute pneumonia, from which it is also differentiated by the absence of dyspnœa, or appropriate thermometrical record. The environment separates it from chronic phthisis.

The condition itself is one of engorgement, or intense congestion of pulmonary substance; it is important to distinguish it from true pneumonia, in view of prognosis and treatment. Associated with the above respiratory murmur, there is moderate dullness on percussion, and fine crepitant or sub-crepitant râles may be heard, *bilaterally*, pneumonia being usually unilateral.

Hemorrhagic Infarction produces increased density and lessened elasticity of the pulmonary substance, and can be recognized by the physical signs applied on general principles.

Atelectasis occurs chiefly in connection with catarrhal pneumonia and some forms of bronchitis, occurring in children in whom nutrition is imperfect and the chest development faulty. It is also produced by the pressure from enlargement of the mediastinal glands, or by aneurismal tumors of the aorta associated with more or less general arterial atheroma. In these latter cases the collapse is associated with nutritive changes in the lungs, upon the exact nature of which there is much controversy. It is a most important fact, that in children suffering from bronchitis, atelectasis may occur in

a few hours, and, under favorable circumstances, disappear as rapidly. The physical signs have been noted elsewhere; but, in children, the advent of atelectasis is associated with less dullness on percussion than pneumonia, and the bronchial breathing is more distant. United with these physical signs, we have the clinical environment of general capillary bronchitis.

In closing this chapter the opportunity appears as suitable as any to introduce some general observations.

Pneumonic exudation into the upper lobe may be so complete as to obliterate the bronchial tube leading to an apex, and cut off the entrance of air into the bronchial tree, so as completely to abolish the respiratory murmur and the respiratory movement. Pressure exerted on the bronchial tube by the distended aorta, in cases of aortic aneurism, is capable of reducing materially the respiratory movement by its pressure, either on the bronchial tubes or on the pulmonary structure itself. It has been just now said that the distention of the thorax is chiefly maintained by the contained air; but there is also a thoracic resiliency which assists the expansion and retraction of the thorax; it is by this prerogative that the lower portion of the chest usually presents an unaltered contour, even in advanced consolidations. Quite the opposite condition, for the most part, prevails in pleurisies of chronic type, in which pleural adhesions are almost invariable, and these exert a retractive force sufficient to overcome the eccentric resilience. This is so unvarying that, with propriety, a rule may be formulated endorsing the statement that retractions of the apices indicate phthisis, while retractions of one or both of the bases indicate chronic pleurisy.

CHAPTER VI.

INDEPENDENT MODIFICATIONS OF RESPIRATORY RHYTHM.

We have already spoken of modifications of rhythm in early phthisis, and shall allude subsequently to its important modifications in diseases of the pleural membranes. We divide the pathological conditions influencing respiratory rhythm into two classes. (*a*) Modifications synchronous with cardiac rhythm. (*b*) Modifications simply of respiratory rhythm. The first class includes pressure of a large heart upon the lung, or pressure of an aneurism upon the bronchial tubes or lungs. In addition, pleuro-pericardial adhesions will often produce a noticeable interruption of rhythm. In class (*b*) the pressure of a tumor in the mediastinum on the bronchial tubes, tumors of the lung, hydatid, specific, cancerous,—are all most liable to influence the rhythm. Inflammatory or other fluids accumulated in the bronchial tubes, as blood, or the serum in cases of œdema of the lungs, shorten respiratory rhythm.

The *quality* and *pitch* of the respiratory murmur is influenced by the principles previously laid down, and the modifications of the vesicular murmur will accord with the physical condition of the tissues in point of density, elasticity and tension. Whatever be the quality or pitch of the respiratory murmur, it is often very feeble. In cases of mediastinal pressure on the bronchial tree, the bronchial breathing may be high pitched and whistling in quality, in proportion to the lessening of its calibre, or, on the other hand, a bronchial tube may be compressed and its lumen positively obliterated. Caution must be exercised in the latter case, not to mistake the loud murmur in an opposite bronchial tube to be patho-

logical, a mistake easily made since the tissues concerned are deeply situated.

The spot at which to auscult for evidence of pressure on the bronchial tubes is over the second dorsal vertebra, but all the other methods of physical diagnosis may be invoked, and conducted under general principles. Modifications of the respiratory rhythm occur during *neuralgias* of the intercostal nerves or muscles, and during angina pectoris.

To make a differential diagnosis between the intercostal neuralgias and pleural or pulmonary disease, we note that the neuralgias are associated not only with pain, but also with tenderness on pressure, the pulmonary processes by pain alone; neuralgias are also unattended by fever. For the diagnosis of angina pectoris we refer to its appropriate section.

Conditions within the abdominal cavity, dropsies, tympanites, enlargement of the viscera, morbid growths, are capable of seriously modifying the pitch, the quality and the rhythm of the respiratory murmur.

CHAPTER VII.

ADVENTITIOUS CAVITIES IN THE PULMONARY SUBSTANCE.

The formation of cavities in the lungs is a sequence in the course of the various forms of phthisis. Subacute inflammations, pneumonia, and gangrene are all elements in the development of cavities. The processes by which these losses of substance occur may be briefly stated—(*a*) by a slow or rapid process of fatty degeneration, followed by ulceration; (*b*) as the result of chronic bronchitis, and softening of bronchial tissue, with subsequent yielding to traction from without; for instance, in broncho-pneumonia or fibroid phthisis; (*c*) abscesses as a sequence of acute lobar pneumonia, following hepatization or purulent infiltration; (*d*) as the direct result of gangrene, itself the immediate consequence of wounds of the lungs, or blood poisoning, or of emboli.*

There are two locations for vomicæ, the pulmonary and the bronchial tissue. In the pulmonary substance they are situated most frequently at the apices, but may occur at the base of the lungs. They are usually small, though they vary in size from a walnut to an orange, or even larger; or there may be multiple small sacs opening into a bronchial tube, like so many blind pouches. In the latter case, the appearance of a section of the lung resembles a large honeycomb. A single cavity may also terminate in a bronchial tube. Cavities are usually situated more or less superficially. The walls of the deeper cavities are composed of solidified pulmonary tissue, or they may be made up of patches of uninvolved tissue, or

* Local gangrene, on a small scale, occurs sometimes around cavities in the lungs, or in the bronchial tubes, and may give rise to *temporary fetor of breath*, but is not likely to lead to fallacious inferences, chiefly because of its temporary character and the absence of permanent concomitant symptoms.

tissue in a state of vesicular emphysema. It is this fact which renders auscultation a valuable means of recognizing deeply situated cavities which would be passed over unnoticed if we relied only upon percussion. By observing the quality of voice and respiratory murmur mistakes can usually be avoided.

Superficial cavities are frequently attached to the chest walls by adhesive inflammation between the visceral and costal pleura. This inflammation is developed in the pleura by contiguity of inflammation in the lung. The presence or absence of this adhesion of the pleura is a factor of great importance in physical diagnosis, since it prevents the pulmonary tissue from collapsing on expiration, thus maintaining the walls of a cavity in a state of more or less distention. There may be many different degrees of thickness of the walls of superficial cavities. They may be so thin as to be composed of the pulmonary pleura only. Adhesion between the visceral and costal pleura prevents the development of pneumothorax by rupture of the walls of a cavity, a possible accident when the cavity walls are thin (see Pneumothorax).

Bronchial Dilatation. When cavities occur in the bronchial tissue, dilatation takes place in two forms: either the tubes are uniformly dilated, like the fingers of a glove, or else they form cavities by undergoing saccular enlargement. The cavities are usually situated at the middle or lower third of the chest, and may be strung in a row following the distribution of the bronchial tubes. It consequently follows that these cavities are recognized anteriorly as located near the second and third costo-sternal junction, or posteriorly somewhere in the near vicinity of the third dorsal vertebra, at which point the bronchial tubes are superficially situated. The cavities may be unilateral or bilateral, according as they accompany chronic bronchitis or fibroid phthisis. The percussion resonance is commonly not so characteristic as over an ordinary phthisical cavity. Frequently for years the

general health is almost unimpaired, and it is never impaired proportionately to the degree indicated by the physical signs. There is no hemorrhage or night sweats, and emaciation is not a pronounced symptom. The same physical signs persist for months or years unchanged, contrary to the history of most phthisical cavities, which continually alter with the advancing malady. The expectoration of bronchial dilatation is more abundant, fluid, and purulent, than in catarrhal phthisis, and is usually brought up in morning or evening by the cupful; it is not a constant spitting of nummular sputum, as in true consumption (*see* Fibroid Phthisis). In chronic cases the expectoration may become so fetid as to generate suspicions of gangrene; the cough is harrassing, but is often relieved if the bronchial cavity is thoroughly emptied. These cases are often mistaken for true consumption, whereas their prognosis is very much more favorable.

Cavities of the Nature of Abscesses. Cavities due to pulmonary abscess are usually situated in the base of one lung, the other being frequently healthy. The pathology of these cavities, with its coincident history, is not that of phthisis. This history is either recovery by contraction (especially after wounds), or more frequently the abscess grows larger and larger till the entire lung may be destroyed, in this respect resembling phthisical cavities. When death occurs it is by exhaustion and hectic; where recovery takes place it is by free opening externally or internally, and evacuation of the contents. At times, the small amount of constitutional disturbance, slight degree of emaciation, the good pulse, easy breathing, slight cough, and comparatively healthy complexion are in noticeable contrast with the physical signs.

Phthisical Cavities are commonly situated in the upper lobes of one or both lungs; if in one lung only, the lung is usually more or less infiltrated by phthisical change. Usually

both sides of the lungs are involved, although the process is furthest advanced in one of them. They are associated with symptoms which include as prominent features, gradual emaciation, persistent loss of weight, by reason of mal-assimilation of food, more or less frequent hemorrhage, and hectic, frequent pulse, hacking, intermittent cough, nummular sputum, expectorated in varying amounts throughout the twenty-four hours; not periodically, as in bronchial dilatations; nor inaugurated by a gush of pus and mucous, as in abscess.

Diagnosis of Cavities. *Percussion.* Percussion is useful in proportion as the cavity is near the surface. In order to appreciate the percussion note of cavities we must have some standard of comparison ; this standard is furnished by the percussion note of the small intestine. This reverberation will be tympanitic in quality, and of high pitch as compared with other pulmonary percussion resonance, or with the tympanitic note of the stomach. If the cavity is deeply situated, the sound may be simply a tubular note, without hollowness, in which case the diagnosis of cavity rests chiefly on auscultation. If the cavity be very superficial, and possess thin walls, the hollow quality will become very pronounced, the pitch very high. The expression amphoric tympany is used to describe this resonance. Amphoric is a word taken from amphoræ, a jar, and represents the resonance of a thin glass vase, a very hollow, high-pitched sound. This hollow percussion is especially marked if the walls of the cavities are united by adhesion to the thoracic pleura. Between the tubular tympanitic, and the amphoric, every gradation of pitch will be found, but the quality of resonance is always hollow, empty or cavernous. The sounds are high-pitched in proportion to the nearness of the cavity to the surface, and the thickness or thinness of the chest walls. If a cavity is located at the apex of the lung, or if the chest walls are very thin, the abnormal resonance may be pronounced.

Cracked-pot Resonance is a term adjectively used to describe a jingle or broken quality imparted to the hollow resonance usually indicative of cavity. The sound may be imitated by striking the clasped hands on the knee; sometimes the sound is as broken as the resonance of a cracked goblet. Two sorts of cavities yield this percussion, the single superficial cavity communicating with a bronchial tube, or a series of small cavities terminating in one opening leading into a bronchial tube. In this latter case, the illustration just furnished gives an explanation of the resonance, for just as the air is expressed through the fingers when struck against the knees, so the air is driven by the shock of percussion out of the cavities, or loculi, and into a bronchial tube. One of the striking peculiarities of the sound is, that the cracked-pot quality is a transient attribute, in many cases, of this phase of tympanitic resonance. The jingle has seemed to the writer due to the transit of the air through fluids in the bronchial tubes entering the cavity. Coughing or expectoration will often abrogate the quality, which half an hour later may return. It is possible that the sound may be developed by some conditions of cavity wall not already noted. However this may be, the essential point is that there must be an open bronchial tube leading into a cavity.

"A cracked-pot sound is possible as a glottidean sound produced by the rapid irregular vibration of the vocal cords." (Wintrich.) This explanation may be applied to the source of cracked-pot sounds in children, in whom the chest walls are thin and yielding, and the air can be readily expelled from the bronchial tubes by percussion. In a few adults with healthy lungs, but thin, yielding chests, a cracked-pot sound is possible, but the quality of the sound is never so hollow as the cracked-pot sound over a true cavity.

There is a peculiarity of percussion stroke designed to give the most favorable expression to cracked-pot resonance.

The blow should be heavier than usual, the plexor being allowed to remain an instant on the finger used as a pleximeter. This implies a deliberate percussion, and it is also well to cause the patient to rest the back against the wall, or a door, before percussion is attempted. Finally, direct the patient to open the mouth, the ear of the listener being placed as close to the mouth as possible (especially if the sound is not immediately distinct when the mouth is closed), for by this means the air tubes act as telephones, transmitting the most feeble cracked-pot or hollow resonance to the ear.

Auscultation of Cavities. We cannot readily recognize by auscultation cavities which are smaller than a walnut. The variation from the bronchial or tubular respiration, consists essentially in the change from a concentrated high-pitched sound, to some tone of hollowness. The hollowness of respiratory murmur is termed cavernous breathing. It differs from the tubular sound in that it has a low-pitched tone or quality which is especially marked on expiration, while the tubular or bronchial sound is high pitched. The reason for this is that since the cavity which yields this sound is deeply situated in the pulmonary substance, its walls can expand on inspiration and collapse on expiration, instead of being unyielding, as the bronchial tissue. Superficial cavities usually have tense walls, which do not collapse on expiration ; thus the echo is more high-pitched, and the hollow respiration is termed amphoric, or a high-pitched empty sound. It is audible on inspiration, but is usually best marked on expiration. There are many grades of breathing between the amphoric and the cavernous; a frequent change being the hollow-tubular tone which is recognized over small cavities surrounded by dense consolidation. It is also heard in bronchiectatic cavities.

Modifications of Respiratory Murmur Due to Situation of Cavities. Cavities situated near the apex anteriorly usually

yield a typical amphoric respiratory murmur, while the same sized cavity situated in the scapular region may, from the density and thickness of the external tissue at that situation, yield a cavernous breathing. If the rhythm of the respiratory murmur be normally maintained, these sounds are typically developed, but feeble inspiration may mask them completely. It is of utmost importance that full inflation of the chest shall occur: this is to be secured sometimes by causing the patient to cough, and sometimes by desiring a series of quick but short inspirations. If the tissues covering the chest are thick the hollow respiration is correspondingly muffled.

Vocal Resonance over Cavities. The vocal resonance is a most valuable aid in the recognition of deeply seated cavities, especially those developed in the scapular region. Instead of the concentrated bronchophony which is heard over areas of consolidation, the bronchophony acquires a hollow quality which distinguishes it from other varieties of vocal resonance. Bronchial dilatations may be mistaken for pulmonary cavities, if reliance is placed on this means of diagnosis alone.

Pectoriloquy. When the phonation is repeated until the words spoken are heard distinctly articulated over the cavity, to this resonance the term pectoriloquy is applied. It is developed by two species of phonation, audible and whispered. The first is too resonant and intense a sound to be readily discriminated from bronchial or exaggerated vocal resonance, but whispered pectoriloquy is very diagnostic. In order that the pectoriloquy shall indicate the presence of a pulmonary cavity it must have a hollow quality.

Palpation over Cavities. The results from this method of diagnosis are negative, since the air within a cavity diffuses the pulses of the air so that they cannot be recognized by the sense of touch.

Inspection over Cavities can, at most, show a pronounced retraction of the chest walls.

CHAPTER VIII.

PULMONARY EMPHYSEMA.

The tenor of our remarks has been designed to encourage reflection upon the effects of departures from the physiological pulmonary condition occasioned by widely different causes. But there are certain conditions to which special reference must be made; one of these seems in place at this point in our study.

Pathological Anatomy and Etiology of Emphysema. The morbid process of general pulmonary emphysema consists in a progressive dilatation of the air sacs, with more or less destruction of their septa, and a consequent associated increase in the bulk of the lung, or hypertrophy. It has received the designation substantive, because there is an actual change in the substance of the lung. The atrophy of the septa and dilatation of the air vesicles results in diminution of the aerating surface for the ramifications of the pulmonary capillaries, consequently there is an interference with the flow of blood through the lungs, and the many consequences of venous repletion ensue. From an etiological standpoint emphysema is frequently the sequel of chronic bronchitis, or recurrent attacks of asthma, and it is often developed in those who have played for years on wind instruments. These various causes result, in one way or another, in the gradual increase of the residual air in the lungs, on account of the difficulty in performing expiration. It is essentially a chronic malady, but in its course subacute attacks of bronchitis occur, which augment the difficulty of respiration. The bronchitis results in more or less blocking of the bronchial tubes with cells and mucous. The air is drawn into the chest by the power of the inspiratory muscles and the thoracic

resiliency, but it cannot so easily pass out again. It is this which, in the course of the disease, so much annoys the patient; his whole effort being to force the air out of the pulmonary tissue. The effects of emphysema are manifested in the local tissue changes and in the general symptomatology.

The obliteration of the blood vessels results in a sense of constriction in the chest, the dyspnœa, the anxious look, the bluish lip, characteristic of emphysematous patients, and dilatation or dilated hypertrophy of the right side of the heart ensues.

The manifold effects of deficient oxidation are too numerous to mention, but very prominent is the lesion of fatty degeneration of the tissues. The imperfect circulation in the chylopoetic system results in imperfect assimilation, indigestion, tympanites, and all along the lapse of years before the patient succumbs, is a liability to repeated attacks of bronchitis, from the continued congestion of the mucous membrane due to repletion of the radicles of the pulmonary artery. In the end transient dropsies may indicate the overloaded state of the right heart. The characteristic physical signs are bilateral, unless there are chronic lesions in one lung, such as to forbid emphysematous change. The increase in bulk of the lung results in a displacement of the liver downward to a variable distance; the heart is displaced downward, and the enlargement of the right ventricle increases the size of the area of cardiac dullness, to the right of the sternum, evidenced by pulsation of the right ventricle in the epigastrium. Displacements of the stomach and spleen are of less importance, but are interesting in view of the tympanites and indigestion of emphysematous patients.

Percussion in Emphysema results in a widespread tympany, of a pitch which is variable and dependent on the degree of emphysema, and the consequent loss of pulmonary elasticity and increase of pulmonary tension. It is always a

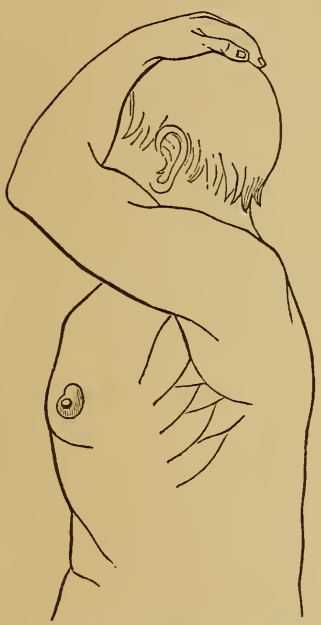
lower-pitched note than can be developed over pulmonary cavities, since the tension is never so great, nor is the elasticity as completely destroyed. It is a higher-pitched tympany than the stomach, since the size of the cavities containing the air are smaller, and the vibration of the pulses of air necessarily shorter. The increasing bulk of lung causes them to bulge above the clavicles, especially during coughing or powerful expiration and the heart and liver are enveloped, so that their areas of dullness are obliterated and replaced by tympanitic percussion; but the tympany will be more high pitched than elsewhere.

Auscultation in Emphysema. The quality of the respiratory murmur is variable, proportionate to the amount of hypertrophied tissue. If the emphysema is general and pronounced, the respiratory murmur is lost. Since the vesicular structure is permanently distended with residual air, we do not hear the vesicular part of inspiration due to gradual vesicular expansion, as in health. We do not hear vesicular collapse in physiological states; in emphysema, on account of the loss of elasticity, there is but little vesicular collapse, and since the bronchial tissue is muffled, by the distended vesicular tissue, the respiratory murmur is extremely feeble. The diminished pulmonary elasticity manifests itself chiefly by changes in rhythm. Inspiration is audible as a short sound, but in reality it is prolonged; expiration is notably prolonged, possibly somewhat interrupted. A most important fact relates to the pitch of expiration, which is low, while in pulmonary consolidation the pitch is high. Frequently, however, the amount of vesicular emphysema is relatively small, while the antecedent bronchitis has wrought more or less extensive bronchial alteration, dilatation, and thickening. In these cases the evidence by auscultation reveals much more of a broncho-vesicular or harsh respiratory murmur than previously described. Cases of emphysema are frequently associated with râles, more or less

numerous, from inter-current bronchitis, to which allusion will be made subsequently.*

Vocal resonance and fremitus in emphysema are negative; the diffusion of the voice yields a distant vocal resonance of normal quality. In the cases in which there has been prior bronchitis, the vocal resonance is harsh. Vocal fremitus is diffused, through the increased bulk of the lung, and is therefore, much diminished, nearly as much as in pleural effusions, but the associated physical signs separate the condition very widely from such diseases.

Inspection and Mensuration in Emphysema. By these combined methods we study modifications which belong to the natural history of emphysema. 1st. The bilateral enlargement of the chest in general substantive emphysema. The change of contour in the chest of typical cases is so marked that the impression of the tendency toward dilatation is obvious.



Showing increase in ant. post. diameter of the chest in Emphysema.

Full inspiration, after overcoming the obstructions, inflates the chest to its utmost capacity. This, of course, elevates the ribs, and causes a transition of the elliptical into the circular form of thorax. Its antero-posterior diameter is notably increased; the vertical diameter is lessened. In a well-marked case of emphysema the thorax is in a state of distention

* Emphysemas preceded by bronchitis are often comprehended in the group of broncho-pneumonia, but in true emphysema there is rarely a tendency to chronic consolidation, although death may occur from pneumonia.

beyond that which would have been produced during health by the deepest possible inspiration. Then comes expiration, associated with powerful diaphragmatic contraction; the lower ribs, which are free to move, are drawn inward to such a degree that there is a distinct sulcus furrowing each side of the chest, running obliquely forward; or the entire inferior portions of both sides of the chest are drawn inward, so that the upper portion having become circular, the whole chest assumes an appearance not unlike a barrel. But still further, the upper part of the sternum being less free to move than the inferior part, there is a species of arching forward of the sternum, and a sinking inward of the lower portions of the bone. To give fullest scope to inspiration, the spine arches forward, sometimes so as to render the sternum nearly vertical, and the front of the chest appears flat; the shoulders follow the spine, and thus a stooping posture is encouraged.

Respiratory Movements in Emphysema. The resiliency of the chest in marked cases is so nearly nil that the breathing is thrown on the upper part of the lung, and respiration becomes superior thoracic, but on the whole, the breathing in a typical case is so feeble that the chest walls scarcely move. There may be at times some diaphragmatic movement. In such cases the sternum and ribs, which have become united by ossification of the costal cartilages, move up and down as one piece, during the expansion of inspiration, while the inferior ribs are drawn inward during the descent of the diaphragm, to expand again on expiration. There is also a noticeable movement of the intercostal spaces. In extreme cases, as I have said, respiration is so feeble that the interspaces are on a level during the period of pause, and the pulmonary elasticity is abrogated. It is in these cases only that the tissues sometimes bulge on inspiration. In less marked cases the eccentric thoracic resilience is opposed by some remaining pulmonary elasticity, and while the chest walls are carried outward, the intercostal

tissues are drawn inward during inspiration, and bulge outward during expiration.*

Once again, we cannot discover by inspection the apex beat in its physiological position. On the contrary, the impulse of the heart is observed in or near the epigastrium; it is not an apex beat, but is the pulsation caused by the thud of the enlarged right ventricle.†

Modifications of the extreme emphysematous change are frequent, and occur in many cases of bronchitis. The departure from a normal standard toward the emphysematous type of chest furnishes in certain cases of phthisis a key to the influence of bronchitis in its etiology, and gives valuable hints in therapeutics.

Mensuration in emphysema simply confirms the results of inspection.

2d. *The pigeon and rachitic change*, referred to in the section on inspection of the normal chest, is often associated with more or less emphysema, usually consequent on the bronchitis, due to the effects of imperfect pulmonary expansion to which the deformity renders them liable. Inspection at once recognizes these deformities, and directs attention to

* Intercostal tissues move inward. *See* also Pleural effusions—Pressure on bronchial tubes; also obstruction of bronchial tubes, as in capillary bronchitis, with atelectasis in children, also in a few cases of chronic fibroid phthisis; also pleural adhesions.

† Causes of epigastric pulsation, in order of frequency:—

1st. Enlarged right ventricle.

2d. Pulsation of abdominal aneurism.

3d. Aortic pulsation communicated to tumors in the abdominal cavity.

4th. Enlarged and pulsating liver, in connection with disease of mitral or tricuspid valves.

5th. Tremor of normal tissues, transmitted from the aorta, if there be some tympanitic distention of abdominal viscera.

6th. Displaced left ventricle in cases of acute or chronic pleurisy.

For dyspnœa in emphysema see Cardiac asthma.

incident pathological changes, which in turn reflect light upon the results of auscultation and percussion.

3d. *Unilateral Enlargement. Inspection.* The etiology of this condition usually differs from the preceding, in the fact that the emphysema is due to vicarious pulmonary activity. Supplemental action of the lung, or vicarious emphysema, is an over distention of the vesicles, comparable to the stretching of the fingers of an old glove, and the pulmonary tissue is capable of resuming its normal condition. If, however, the vicarious activity persists indefinitely, the substantive changes already described are developed. The causes of unilateral enlargement are those which demand supplemental activity of one lung, through disablement of its fellow.*

Mensuration in Unilateral Enlargement. Circumferential measurements of the one or both sides are obviously capable of yielding results; but when one side is to be compared with the other by measurement, remember "that considerable increase in the sectional area of the chest may occur, and the length of the periphery remain the same by the passage of the elliptical form into the circular, and next, that the displacement of the mediastinum, which accompanies unilateral enlargement, thrusts the heart into the unaffected side. Add this consideration too, that the walls of the healthy side must follow the antero-posterior projection of the diseased side, and then it will be plain why, as a matter of fact, the perimeter of the expanded side often measures very little more, nay, even less, than that of the side which is not diseased." A tracing on paper, by indicating not merely circumference, but also shape, affords us the true means of recording the amount of unilateral enlargement.

Auscultation in Unilateral Enlargement. Exaggerated respi-

* In chronic pulmonary diseases, Louis tells us, the upper lobe of the right lung or the lower lobe of the left are the parts most frequently emphysematous, and present visible local bulging.

ration is the type, often of such intensity that it simulates the bronchial. It is, however, never so concentrated or high pitched as bronchial breathing, indicating consolidation.

Diagnosis of Bilateral Emphysema. Pneumothorax is probably the condition most apt to be mistaken for emphysema; bilateral enlargement of the chest from this cause would be incompatible with life. (For further details see chapter on Pneumothorax.) In diagnosis of unilateral emphysema, attention should be given to the morbid processes in the opposite side of the chest, especially pleural effusion.

Atrophic Emphysema. Allied to the preceding group is another form of emphysema, in which that pathological state is ultimately linked with atrophy of all the pulmonary tissues, and with atrophy and fatty degeneration, not only in the lungs, but as a widespread degenerative change, including especially fatty degeneration of the heart muscle. We find in these cases, atheroma of the arteries, arcus senilis, defective nutrition, loss of appetite, with deranged functional activity of the liver, loss of sleep, dyspnœa, all developed largely in consequence of fatty degeneration of the heart, and ultimately associated with its symptomatology.* We have cough and evidences of bronchitis dependent on the enfeebled circulatory power, and sometimes areas of positive congestion. The ensemble is almost exclusively seen in middle or advanced life, but sometimes these changes occur in those in whom alcoholism, syphilis, etc., have engendered in early life these degenerations, commonly the dealings of the hand of time. It also occurs not rarely as a consequence of the arrest of previous pulmonary phthisis. The pathology implies an atrophy of some of the vesicular tissue, leaving dilated emphysematous air sacs interspersed among the normal, and those in which partial atrophy of inter-vesicular substance and intra-vesicular septa has taken place. The pathological

* *See* Symptoms of Fatty Heart.

picture includes, perhaps, a small amount of cirrhosis, sometimes more or less vesicular consolidation, but always dilatation and thickening of the bronchial tubes and bronchitis. It occurs as a bilateral, as well as a unilateral process, and inspection is very helpful in diagnosis.

Inspection in atrophic emphysema includes: 1st. A universally flattened chest anteriorly. 2d. It has all the fixed, inexpansible look of emphysema, with the changes in costal cartilages. The sterno-mastoid muscles stand out strongly against the sunken supra-clavicular spaces, unless they are bulged by a forcible expiration. 3d. The diaphragm is depressed; there is epigastric pulsation. 4th. The heart is covered by lung, as in hypertrophic emphysema.

Percussion in atrophic emphysema is tympanitic in quality and more or less high-pitched.

Auscultation in Atrophic Emphyesma. Broncho-vesicular breathing, with a low-pitched expiratory sound, which enables one to exclude processes of consolidation.

Vocal resonance and fremitus are negative.

CHAPTER IX.

RALES. ACUTE, CHRONIC AND CAPILLARY BRONCHITIS. ŒDEMA. HYPOSTATIC CONGESTION.

Râles are adventitious or new sounds, which are developed in the pulmonary tissues in one of four ways, and can be classified into two groups.

Group. I. Râles.

(*a*) By the transit of air through fluid in the bronchial tissues, or pathological excavations in the pulmonary tissue.

(*b*) By the transit of air through bronchial tissue, in which the natural calibre of the tubes is diminished.

(*c*) By the entrance of air into the vesicles. (Infrequent.)

Group II. Frictions. Sometimes called râles. (*d*) By the alterations of the surfaces of tissues normally smooth, but which are pathologically roughened, for example, the pleural or pericardial surfaces.

The quality of the râles is the single basis of the classification. In both groups the sounds may be defined as cracklings, as well as râles, but in group second the râles possess a distinct, rubbing quality, so that they are termed frictions. In both groups the sounds are designated by the terms, dry and moist râles or friction sounds, because of the impression they make on the ear. All moist râles developed in the bronchial tissues, are also described by the adjectives large, small, and intermediate-sized râles.

Another cardinal condition for the development of râles is, that the more perfectly respiration is carried on, the more satisfactorily clear will the sounds become. It is a common observation, that on quiet (often feeble) respiration, no adventitious sound can be heard, but as soon as deep respiration is attempted, at once the râles become plainly audible.

Taking up in the first class, division (*a*) or moist râles developed in the bronchial tissue, it is plain that the generic term fluid, may stand for mucous, for pus, for blood, or any other liquid ; at the same time the quality of the liquid has to do with the size of the râle, which is smaller, the more tenacious the fluid. The size of the râle also depends on the location in the bronchial tree at which it is generated; the larger the tube the larger the râle. When there are cavities developed in the pulmonary substance, the transit of the air through the fluid therein, creates a large, moist sound, comparable to a bubble, or jingle, which may have an amphoric echo. It is precisely upon this simple plan that the nomenclature of moist râles has been more or less minutely attempted. The sequel of efforts at inclusive designation has led to much complication. Regard all nomenclature as tentative ; christen the râles on the basis just stated ; using the word mucous as a generic term sanctioned and defined by precedent, as inclusive of the many varieties of liquid-occasioning râles.

Simply for convenience of description we would suggest the following classification :—

Mucous râles, large } or gurgling, may have amphoric echo.
" " bubbling }
" " small.
" " very small.
" " crackling or subcrepitant.
" " crepitant.

Amphoric râles are to be classed as indicative of large air-containing cavities, possessing thin walls containing fluid, connected with an air tube. A metallic tinkle is a bell-like mucous sound, which can be artificially developed by striking sharply with a pin a metallic glass or porcelain cup. It is a sound developed by the splashing of particles of fluid in a cavity which possesses tense thin walls and is freely supplied by air.

It can also arise from the splash of mucous falling into a cavity. Tense walls pertain to conditions which are found in pneumothorax, when the opening into the lung is patulous, or in a very large cavity with thin walls at the top of the lung. It is possible, but extremely unlikely, to hear a metallic note imparted to a large mucous râle developed in the bronchial tubes.

In class (*b*) we find essentially dry râles, called,

Sibilant or high-pitched, hissing râles,

Sonorous or low-pitched, snoring râles,

according to the size of the air-passage in the bronchial tubes in which they are generated. These râles are developed by the transit of air through the bronchial tubes, which are narrowed by a swelling of their lining mucous membrane, or by a large plug of tenacious mucous. This explains the fact that they are sometimes displaced by coughing. The sibilant râles have all grades of quality; whistling, cooing, hissing, piping, sawing, fluttering, all are terms used for their designation, according to the fancy and verbosity of the observer. Dry râles frequently, and moist râles occasionally, give rise to a fremitus which is not only audible, but which can be felt. In some cases of chronic bronchitis, moist râles give rise to a fremitus, but the many marked evidences of bronchitis prevents any confusion.

Class (*c*). These râles are vesicular, caused by the severing of the walls of the vesicles rendered adherent by small amounts of adhesive fluid. They are called crepitant râles, a sound which resembles the clicking caused by the lighting of a chandelier by electricity. It can be imitated by the rustling of the finest silk, or the rubbing of the hair between the fingers, close to the ear, or by separating the thumb and finger rendered adherent by mucilage. The sounds are both dry and moist. The dry crepitant râle is almost limited to the incipient stage of croupous pneumonia, when exudation

is just beginning to be poured out into the vesicles. It is characteristic of this râle to be developed all over the site of inflammation, and it will be remembered that an entire lobe, or at least a large section, is invaded in this disease.

The moist crepitant râle is sometimes heard in conditions of œdema, at its very outset, but the sound is bilateral, and soon becomes a subcrepitant râle. Moreover, it is associated with the general conditions predisposing to œdema of the lungs.

Group II (*d*). The friction râle is dry or moist, according to the stage of the pleurisy in which it is developed. The dry friction râle is, in quality, a rubbing sound, developed between the pleural or pericardial surfaces. It has been compared to the creaking of a new leather shoe; it is nearly always a grazing, or tearing, or grating sound. The moist friction is developed when serous surfaces, whether pleural or pericardial, are approximated, and rub against one another, especially in the early stages of inflammation. It is a very fine cracking sound, often acoustically indistinguishable from the fine, moist, subcrepitant râle within the lung.

These râles manifestly occur both with respiratory and cardiac rhythm. The true frictions are preëminently superficial sounds, and the respiratory murmur, with or without râles, can, with practice, be determined to be a beneath sound, developed in the deeper tissues. They are essentially grazing sounds, of come-and-go quality, not commencing with inspiration but toward its close, and ending before complete expiration. The rubbing, or harsh quality of the creaking or grating varieties of friction are thus easily recognizable; not so the finer forms.

The finer forms of friction are not influenced by the succussion of the act of coughing. They are developed usually over the inferior portions of the chest, anteriorly or posteriorly, and frequently over a limited space. They can

often be abolished by the following manœuvre: when the chest is fixed, especially at the lower two-thirds, by the embrace of an assistant, or even by tightly-drawn adhesive plaster, and the ear or the stethoscope is applied over the doubtful sounds, they will be found to have disappeared, if of pleural origin; but to be still discernible if bronchial râles. Further, cause the patient to incline to the opposite side to that diseased, and place the hand of the diseased side on the head; this puts the pleura in a state of tension, and often will obliterate a friction râle.

The methods most available in the diagnosis between pleural and intra-pulmonary râles are, 1st, the fixation of chest as described. 2d, the influence of coughing. This will disperse or alter the size and location of intra-pulmonary râles, but a friction sound will be unchanged.* On the other hand, the grazing quality of the pleural friction, and its association with but a portion of either respiratory movement, even on full respiration, will in most cases enable us to discriminate. 3d, the ear must be trained to observe sounds generated on different planes of tissue. 4th, the association of the other means of diagnosis of acute or chronic pleurisy, or pericardial diseases, must be invoked. 5th, especially note the associations of these râles with the limitation of expansion observed by inspection in acute pleurisy, or the retractions observed in chronic pleurisy. 6th, the etiology of pleurisies or pericarditis must be studied.

Diagnosis between friction râles of *respiratory* and *cardiac rhythm* is considered under the section on diseases of pleura and pericardium. Having defined the attributes of râles, it is next in place to study the conditions under which they are developed.

In all the varieties of subacute or chronic inflammations of

* Exceptionally, friction râles may be temporarily increased during the deep breathing which follows the act of coughing.

the lungs, râles are developed as the result of the softening of the inflammatory new formations, or as the result of intercurrent bronchitis, acute or chronic. Acoustically, it is impossible at times to decide whether the râles are the harbingers of the softening resulting from inflammatory action, or are the expression of an intercurrent bronchitis. In making differential inferences observe some of the following propositions: If the râles are harbingers of the softening of lung tissue, ere long their etiology will be attested by cavities with gurgling râles, and appropriate auscultation and percussion. Moreover, if râles are due to softening, they will be limited to the areas already designated by auscultation and percussion, as the areas of inflammatory new formation. The higher range of the thermometrical record indicates the softening of the inflammatory new formation, rather than bronchitis. If there are evidences of consolidation, and the râles are small, not very moist, subcrepitant, crackling and localized, they are usually due to local processes rather than general. Sibilant and sonorous râles are frequently heard in the course of phthisis, indicating either an intercurrent bronchitis, with swelling of the mucous membrane, or the plugging of a tube with mucous. When local phthisis exists, bilateral râles may be present, and may be explained by an intercurrent general bronchitis, because the phthisical are very prone to this inflammatory process. Deductions may also be drawn from the history and progress of a case; if the râles disappear under treatment, the presumptive evidence is in favor of their being simply bronchial.

Râles in the Early Diagnosis of Phthisis. In incipient textural modifications in the lung it is well to await the advent of crackling, or some species of moist râle, before deciding that the evidences of percussion and auscultation relegate the tissue modifications to some group of phthisis. In these cases very often these râles can only be excited by the act of coughing.

Râles in Cavities. When these are filled with fluid, naturally all kinds of splashing sounds, from small to large gurgles, can be heard. Similar râles are sometimes audible in bronchorrhœa. The diagnosis of cavities does not depend upon the species of râles heard. If bronchiectatic cavities exist they may be recognized and differentiated by signs already stated.

Râles in Acute Consolidations. Croupous pneumonia—The dry crepitant râle is heard during the initial stages of the process, but *invariably* disappears when consolidation is complete. One must, therefore, auscult very early in the case to secure this râle; but sometimes the area of pneumonia extends, and then crepitant râles precede the involvement of new districts of tissue. (*See* Pleurisy, for diagnosis of pleuro-pneumonia.) Study especially the general symptomatology, with the physical diagnosis, in pneumonia; this is especially important in the aged, in whom the thermometrical record is apt to be low. In these cases the advent of the râles, and the associated signs by auscultation and percussion, must be studied, as well as the immediate results of a physical exploration.

Acute Catarrhal Pneumonia, with its rapidly developed diffused areas of consolidation, is always associated with a widespread development of subcrepitant and mucous râles. It is a disease which not infrequently occurs in children, and is associated with a collapse of neighboring pulmonary districts. (*See* Capillary Bronchitis.) It is associated with descending bronchitis, and often becomes in adults a subacute or chronic process, the forerunner of some form of phthisis, or it may occur in the course of emphysema.

Acute and Chronic Bronchitis. The râles not associated with some of the groups of phthisis or œdema are the râles in which pathological change is solely confined to the bronchial tubes. The division, of course, relates primarily to *acute* and *chronic* bronchitis. In acute bronchitis the

sonorous râles are developed in the larger, the sibilant râles developed in the smaller tubes, and are occasioned by the swelling of the mucous membrane lining the tissues concerned, or the presence of tenacious mucous. After the initial stages of congestion are passed, exudation is more fluid, swelling more or less rapidly subsides, and the various grades of mucous râles appear. The sibilant and sonorous râles become scattered and less numerous, and finally give place altogether to the moist râles. In the chronic phases of this disease the sonorous and sibilant râles may be associated with the mucous, since there may be more or less permanent bronchial thickening or transient plugging of the tubes. Râles indicative of bronchitis are said to be always bilateral. This depends on whether the bronchitis is simple, or is associated with some form of phthisis, or conditions affecting tissues other than the bronchial. The proposition then is: in simple bronchitis, acute or chronic, the râles are bilateral, since râles indicate simply fluid in the bronchial tissue. If other processes are associated, differentiation is based upon the comparative study of symptomatology and physical examination. In acute or chronic bronchitis the further evidences furnished by auscultation are harshness of otherwise normal respiratory murmur, perhaps even broncho-vesicular breathing, which is produced by the changes in the bronchial mucous membrane. There is never the concentration of quality nor the elevation of the pitch indicative of condensation of the pulmonary substance. Percussion is negative. The resonance is normal, or presents such slight elevation of tone as to be insignificant.

Vocal fremitus and resonance in bronchitis are unchanged from normal standards described.

Finally, we again advise a careful study, by percussion and auscultation, of those cases in which the fine mucous râles are present, especially in cases of bronchitis complicating

emphysema, in order to ascertain whether they are indicative merely of bronchitis, or whether they indicate patches of consolidation. Study chiefly the expiratory sound of the respiratory murmur. This is *low* pitched in simple bronchitis, but high pitched in proportion to the amount of consolidation, and its quality becomes concentrated, converting the breathing into the bronchial type.

Capillary Bronchitis is a disease of the two extremes of life, childhood and old age. In children its advent is often so sudden that some laryngeal affection is suggested, such as croup or œdema, but auscultation will prevent this mistake. It is apt to be confounded with and followed by acute catarrhal pneumonia, especially if capillary bronchitis occurs as a complication of measles, pertussis, or chronic catarrhs. In adults, the chief source of error will arise from the confounding of this disease with œdema or hypostatic congestion. When the exudation occupies the finer bronchial tubes, we have two very necessary results, excessive dyspnœa and the finest bronchial sub-crepitant râles; and in these two particulars we find the cardinal difference from ordinary acute bronchitis. There are areas of rather distant bronchial breathing as compared with ordinary consolidation. The percussion is impaired but not very dull. In capillary bronchitis, especially in children, the bronchial tube leading to a district of tissue may be suddenly plugged. Atelectasis may occur, and, in a few hours, the case may assume an aspect of greatly increased gravity. In diagnosis, consider the sudden development of the serious symptoms; especially dyspnœa. By inspection note the absence of full thoracic expansion on the affected side, possibly the increased inward movement of the intercostal tissues at the base, on inspiration. Auscultation is not so helpful. Respiration may be feeble, absent, or distant bronchial. It is sometimes impossible to positively distinguish capillary bronchitis from catarrhal pneumonia. We refer to

the chapter on physical diagnosis of that disease, only adding that capillary bronchitis is attended by a lower thermometrical range than the catarrhal pneumonias, or simple bronchitis. A diagnosis may be made by recognizing the predisposing causes of capillary bronchitis.

Œdema of the Lungs, if acute, is sometimes the result of sudden congestion of the lungs. It is more commonly chronic, and consists of effusion of fluid into the finer bronchial tubes, and the pulmonary tissue generally. It is associated with dropsy of the general cellular tissues or effusion into the serous sacs, as the result of organic disease of heart, kidney, or liver; in frequency, about in the order in which the organs have been named. Its prominent characteristics are fine moist râles, developed in the finest bronchioles, or in the inter-vesicular tissue during inflation of the lung.* There is abundant expectoration of frothy mucous, greatly embarrassed respiration, with intense congestion of the venous system, evidenced by the bluish lip, the feeble or noisy breathing. In acute œdema death speedily terminates the scene. Chronic œdema may be persistent, and so are the symptoms.

Hypostatic Congestion. Subcrepitant râles, associated with hypostatic congestion, due to the exudation into the finer bronchial tubes of serum, are heard bi-laterally, but are differentiated from bronchitis, by their association solely with posterior and basic portions of the lungs, and by their association with special states: from true pneumonia, by their association with these special systemic states, and also, by the absence of sufficient dullness or bronchial breathing.†

* These finer râles are the râles most apt to be confounded with moist pleural frictions.

† If the patient is able to permit respiratory percussion, the greater clearness of sound on percussion during held inspiration will separate hypostatic congestion, and possibly the other conditions, from true consolidation.

A moist crepitant râle can be heard, at times, in states of hypostatic congestion or œdema. The râle is often indistinguishable from a subcrepitant râle developed in the finest bronchioles. There is also one point which will bear reiteration in this place, viz., that pulmonary râles may be heard in inspiration and expiration. In doubtful cases insist on full inflation (often to be secured only by requesting the patient to cough) before giving an opinion. (*See* also Chapter V.)

CHAPTER X.

ASTHMA. ACUTE MILIARY TUBERCULOSIS.

By asthma is meant a spasmodic narrowing of the bronchial tubes, by contraction of their circular muscular fibres. It is idiopathic, without structural pulmonary disease; or symptomatic. Both forms are associated with all varieties of sibilant or sonorous râles, and, after a longer or shorter time, by all sorts of mucous râles. These mucous râles are partly the result of congestion of the bronchial mucous membrane, not indicating bronchitis. If from congestion, they disappear soon after the paroxysm is passed; or they may indicate the association of bronchitis with the spasmodic attack.

The *dyspnœa is expiratory in rhythm.* It is true, there is an obstruction to the entrance of air into the lungs, but in the presence of a case we notice the chest is fully distended. The appropriate expiratory efforts are, however, inefficient, partly from the difficulty in expelling the air, owing to the spasmodic contraction of the bronchial tubes, partly from the lack of stimulation to expiration, on account of imperfect inspiration. The evident result is the apparent exclusively expiratory dyspnœa. The lower parts of the chest are fixed and altogether immovable. Few abdominal respiratory movements occur, and since the diaphragmatic action cannot avail, no such movements are made, and the diaphragm remains depressed. It follows that the respiratory murmur is weakened, inaudible or muffled. The heart sounds also become very weak, indistinct and distant.

It is desirable to group the characteristic features of the attack: 1st, suddenness. It often occurs at night; a patient may wake out of sleep, in a paroxysm of the disease, although a feeling of gradual suffocation may precede the attack. 2d,

intense dyspnœa. The patient desires the windows raised, or walks about gasping for breath. 3d, the skin is cold and moist, of a pallid or bluish hue, showing imperfect aeration of the blood. 4th, the attack may last hours, and disappear suddenly; or it may be prolonged for days, ameliorating during intervals, to again grow worse, at last terminating gradually.

Symptomatic Asthma. This form is sometimes known as cardiac asthma. It is associated with cardiac lesions, such as fatty degeneration, sometimes with valvular disease. It is very commonly interlaced with emphysema and with chronic bronchitis, in which there is often dilatation of the right side of the heart with fatty degeneration.*

Diagnosis of Asthma. Idiopathic asthma frequently results in cardiac changes with emphysema, so that often the conditions of idiopathic and symptomatic asthma are blended. In both there is marked spasmodic paroxysmal breathing. Differentiation must be based upon the associated pathological changes. In symptomatic asthma there is less bronchial spasm, dyspnœa being due to the difficulty in the supply of blood for aeration rather than to the entrance of air into the lungs. A correct appreciation is important, because the treatment of pure spasmodic asthma consists principally in the use of anti-spasmodics: a treatment of symptomatic asthma includes also cardiac and respiratory stimulants.

Œdema and spasm of glottis differ in not being associated with wheezing. Laryngeal paralysis can be detected by the mirror; there is also aphonia, but the stridulous breathing is inspiratory, and there is an absence of adequate chest symptoms.

* True asthmatic seizures may both produce and be produced by a disease of the heart. But what is often called cardiac asthma is not always a spasm of the bronchial tubes. It is usually only a temporary increase of the dyspnœa, dependent upon a decided obstruction to the circulation in the lungs, and not accompanied by wheezing. (See Da Costa, Medical Diagnosis, page 257.)

The pressure from mediastinal tumors, notably aneurisms, cause symptoms similar to asthma by pressure on the pneumogastric nerves or on the bronchial tree. The diagnosis is only thoroughly established by a diagnosis of the nature of the tumor.

Renal Asthma. In asthmatic disorders due to kidney disease, the pulse is very rapid, without cardiac disease to account for it. At the same time the air enters the lungs without obstruction. The asthmatic symptoms are traceable to the difficulty experienced by the blood in reaching the air, due to a spasm of the arterioles of the lungs, instead of an obstruction to the air reaching the blood. The diagnosis is made by exclusion. 1st. Demonstrating the absence of any pleural effusion or other adequate pulmonary cause for the dyspnœa. 2d. By establishing the existence of renal disease, especially those forms most intimately associated with preponderating interstitial changes. As an important auxiliary to establishing the diagnosis of chronic renal disease, note the hypertrophy of the heart, and the accentuation of the second sound.*

Cheyne-Stokes Respiration. A disorder of respiratory rhythm is the so-called "Cheyne-Stokes" respiration. "It consists in at first short, then deeper and more and more labored, respirations, until the paroxysm is at its height; then becoming shorter and more and more shallow until the breathing is suspended. The pause lasts from a quarter of a minute to a minute, when the respiration begins again in the same manner, first faint, then a little stronger, then again subsiding in a descending scale, to end in the same stand-

* Dickinson "states that hypertrophy of the heart occurs in seventy-four per cent. of cases of granular nephritis; page 310 of his Pathology and Treatment of Albuminuria." Hypertrophy may not occur, or it can disappear if the vital activity is reduced below the plane of nutrition capable of resulting in physiological tissue growth.

still. This kind of breathing is a very unfavorable symptom. It is apt to happen when, from some cause, the supply of arterial blood is cut off from the brain or respiratory centre in the medulla. It is rare in diseases of the lungs, much more common in fatty heart, in diseases of the aorta, in tubercular meningitis, in affections implicating the medulla oblongata, and in uræmia." It is also a symptom in cases of apoplexy.

Sansom summarizes the cases in which this sign is observed, as follows:—

"1st. Cases attended with cerebral lesions, viz., cerebral hemorrhage, tumors, uræmia, shock from surgical injury, alcoholism, acute renal disease, tubercular meningitis.

"2d. Cases attended with lesions of the heart or great vessels, viz., fatty degeneration, pericarditis, atheromatous disease of aorta, aortic aneurism, valvular disease (double aortic, mitral stenosis, dilated aorta, coexisting aortic regurgitation and obstruction). Sclerosis of coronary arteries.

"3d. Cases of certain acute febrile diseases, viz., diphtheria (Hütterhenner), typhoid fever (Wharry). A large portion of cases occur in the male sex, and at a period of life over fifty, when degenerative changes are common, the exceptions being in the acute diseases which I have noted. I consider that the initial lesion is paresis of the respiratory centre, and though this paresis *may* be produced by reflex nerve influence, it is usually a direct exhaustion, from cerebral causes."

Acute Miliary Tuberculosis. The general acute eruption of miliary tubercle is distributed in and around the tubular structures in the liver, spleen, bronchial and mesenteric glands. In the lungs the deposit occurs in the sheaths of the arterioles and lymphatic vessels. The membranes of the brain enjoy an immunity in the adult, quite the reverse of the history of similar cases of tuberculosis in the child. The intestines, too, may escape, or even if involved, death happens before much, if any, ulceration occurs. This is an interesting fact, since it explains why diarrhœa is not a symptom; if, however, phthisis end in

general miliary tuberculosis a follicular catarrh or ulceration of the bowels is frequently found quite sufficient to account for a possible diarrhœa. In the lungs, as elsewhere, the translucent gray granulation alone is characteristic of miliary tubercle. The vesicular pulmonary tissue is not consolidated; on the contrary, the lungs bulge with air and do not collapse, showing that the tissue is more condensed than natural, and the lung feels as though infiltrated with fine particles of shot. Tubercle may develop secondarily from some prior centre of inflammation often located in the pleura. There may be in one or other apex a small, or perhaps a large cavity, which antedates by a longer or shorter period the eruption of miliary tuberculosis. The effect of the tubercle deposited in the lungs diminishes the calibre of the arterioles, and the aeration of the blood is less perfect.

Percussion in Acute Miliary Tuberculosis. The percussion is often negative; a vesiculo-tympanitic resonance can be developed over the lungs. Sometimes small areas of dullness occur anteriorly or posteriorly.

Auscultation in Acute Miliary Tuberculosis. Usually an exaggerated respiratory murmur, with feeble, low-pitched expiration is heard, suggesting the type heard in emphysema, or in supplemental action of a healthy lung when its fellow is disabled; nowhere is the breathing bronchial. With this, can be heard throughout the lungs, on inspiration alone, numerous fine, slightly moist râles, similar to the crepitant râles of pneumonia. They also remind one of the râles of capillary bronchitis, but capillary bronchitis is attended with copious expectoration, and is apt to be associated with the chest and heart alterations of emphysema, or the preëxistence of Bright's diseases. Sometimes dry sonorous and sibilant râles can be heard. If there be cavity at an apex it is diagnosticated by appropriate phenomena.

Remarks. The pathology of the disease sheds much light

on the physical signs. The development of the râles just described occurs from the irritation of the bronchial mucous membrane and consequent moderate catarrhal exudation into the finer bronchial tubes. If there are friction râles they will be dry, and perhaps some restriction of the movements of the chest will occur by comparing the sides, but both lungs, nevertheless, expand and retract. Dry pleurisy is especially indicative of a tubercular etiology. In reflecting on the quality of the percussion resonance and respiratory murmur, remember that the vesicles are hyper-distended, as in emphysema. The respiratory act is imperfect, because obstruction to the exit of air is due to the catarrhal secretion in the finer bronchial tubes. The duration of miliary tuberculosis is from six to nine weeks, usually the shorter period. It is an assistance in diagnosis to search for evidence of prior suppurative inflammation. Examine the glands of the neck, note if scrofulosis, or consumption, are hereditary in the family of a patient, for the connection between these conditions and the disease is very intimate.

General Symptoms of Acute Miliary Tuberculosis. There is always dyspnœa, apparently without adequate pulmonary lesions. The cough is not attended with much expectoration; if there be any sputa it is a small amount of frothy mucous. Hemorrhage is not a usual symptom. Sweating is a marked symptom; it is so profuse as often to drench the clothing of the patient and the bed in which he lies. Another valuable symptom is in the dusky hue of the face, which, however, may be flushed as in typhoid pneumonia; at other times it is quite pale. The general surface of the body in the later stages of these cases is dusky, resembling the cadaver after death from a blood disease. Rapidity of the pulse is a salient symptom during the entire course of the disease. It is never, in typical cases, below 120, and may attain a much higher rate. The pulse is small and often thready. Perhaps the

interference with the pulmonary circulation, due to the abundant distribution of the tubercles, may explain this appearance of the body and also the frequency of the pulse. The temperature record is of great value. The range is irregular, usually from 100° F. to 103½° F. In the week before death (for this disease is fatal) the temperature may fall to 97° F., although it may rise again to 101° F.

In cases of general tuberculosis complicating advanced consumption, the physical signs of the antecedent phthisis often mask the none too characteristic physical signs of the presence of the complication. A symptomatology similar to that just described prevails, but with modifications induced by the presence and preponderance of the catarrhal inflammation. The pulse may not be so persistently frequent, unless suppuration has produced exhaustion; even then it is not so characteristic of tubercle, because it is customarily observed in cases of hectic. The temperature curve is often higher, and presents marked daily fluctuations corresponding with those given by Wunderlich and Niemeyer. The elevation of temperature is probably due to the suppuration, while in acute miliary tuberculosis death happens before cheesy degeneration or suppuration occur. In cases of secondary tuberculosis life is often prolonged until cheesy metamorphosis of the tubercle has occurred in addition to the antecedent older cheesy material.

In secondary tuberculosis the dyspnœa is not so great, possibly because the chronic antecedent malady has reduced the systemic demands for oxygenated blood; and although the acute development of tubercle may cause some shortness of breath, it is not so definitely manifested as in the primary disease. The bodies of patients are usually emaciated, while in acute primal miliary tuberculosis there is no emaciation or evidence of wasting. Finally, metallic râles, some species of

cavernous breathing, vocal resonance, or tympanitic percussion, will indicate one or more cavities.

The differential diagnosis in acute miliary tuberculosis consists in a differentiation from typhoid, remittent, or typhus fevers. It is separated from typhoid fever by a rapid pulse early in the case, without the typhoid state, the cyanosis, the physical signs in the lungs, the sweating, the absence of characteristic rise in temperature, and the absence of prodromes from the history. From remittent fever by history and therapeutic tests. From typhus fever: the ensemble is somewhat like many cases of typhus, for in this disease there is the early rapid pulse, and a dusky hue of the skin. The history of the case, a minute analysis of the pulmonary symptoms, the typhus eye, the environment from which the cases are developed, are perhaps the safest indications for diagnosis by exclusion, while the eruption of typhus, if it be characteristically present, is conclusive.

CHAPTER XI.

DIAGNOSIS OF THE LESIONS AFFECTING THE PLEURAL CAVITIES. ACUTE PLEURISY.

In a diagnosis of lesions under the above heading, it is fundamental to consider the pathological conditions affecting the pleural cavities, also the effect of these conditions upon the lungs as air-containing tissues, and upon the methods resorted to as means of diagnosis. There are various forms of pleurisy, both with and without effusion. If there be an effusion it may completely fill the pleural cavity. On the other hand, there are cases of pleurisy in which the effusion is very small—scarcely more than covering the floor of the cavities—between the two we have every possible gradation in amount of effusion. There are two species of inflammation, to which the terms serous and fibrinous have been applied. If the formation be simply serous, the fluid will gradually accumulate until the entire chest is filled. If the effusion be fibrinous, it may spread itself very widely over the surfaces of the pleura, until they are more or less completely coated with flakes or layers of lymph.

Acute pleurisy can terminate—

1st. In cure.

2d. Pleurisy subacute, or chronic, with effusion.

3d. In chronic pleurisy, usually with adhesions.

4th. Empyema.

The pathology of these lesions furnishes a basis for discussion of their diagnosis. In acute pleurisy we are aware that the effusion may be serous or fibrinous. If fibrinous, plastic lymph more or less moistened with serum is distributed over the pleural surface in flakes, which are more or less thickly massed at some points, while at others the pleura may

be normal; there is also always more or less fluid effusion, in which flakes of fibrine float. In the initial stages there is more or less congestion of the pleural vessels, both of the visceral and parietal pleura. The polygonal spaces, which can be seen on the visceral layer of the pleura, represent the bases of the pulmonary lobules; the blood supply of the visceral pleura is intimately connected with the blood vessels of these polygonal spaces. During inflammation vascular projections or vegetations form, over which, if the process proceeds to exudation, the lymph is most thickly coated. But the vascular congestion possesses this further interest, it exhibits the intimate relation of pleural to pulmonary congestions and inflammations. The amount of serous effusion may be large compared with the amount of fibrinous, or the reverse may occur. The aggregated fluid varies from half a pint to a pint and a half; this amount requires several days to accumulate, perhaps a week or more. Occasionally the effusion continues to increase until the pleural cavity is filled. Fibrinous pleurisy is a frequent complication in the history of phthisis, and is sometimes its antecedent. A bilateral pleurisy, indicates a systemic etiology, such as Bright's diseases, pyæmia, gout, rheumatism, typhoid or typhus fevers, scarlatina, measles. The term hydrothorax is applied to any serous effusion into the pleural cavities, but it especially indicates passive effusion resulting from cardiac, renal, or hepatic disease.

The effects of pleurisy upon the lung vary in acute disease, in accordance with the amount of effusion present. If this be moderate, and the pleurisy chiefly fibrinous, the lung will be slightly compressed, its complete inflation will be resisted, and there will be a more or less thickened vascular pleura interposed between the visceral and costal surfaces, so that the air-containing lung is further removed from the ear than is physiological. If there be a pint or more of liquid within

the chest, the lung will be borne upward, and there will be a tendency to compress the pulmonary substance backward. The vesicular tissue is chiefly encroached upon; the bronchial tissues being more resistant, remain nearly as thoroughly patulous and capable of transmitting air as normally. Let us fancy, however, the pleurisy, instead of terminating in resolution and absorption at the above stage, passes on into the stage of large effusion. The effect upon the lung must be considered under a twofold postulate: 1st. The effusion, if very large, may positively compress all the pulmonary tissues, including the bronchial, until the air is absolutely expelled, and the lung itself is flattened into a small space against the vertebral column, occupying mostly the scapular region, or a space sometimes not larger than the palm of an ordinary-sized hand. 2d. The fluid may force the lung upward, so that we may outline it anteriorly, perhaps, so low as the second rib, and the lung occupies a variable area of the scapular region. In either case the lung tissue must be considered as sustaining very abnormal degrees of tension and elasticity. If the bronchial be the only tissue permeable to air, then the pulmonary atmospheric tension must be high, and the elasticity nearly abolished. If some of the vesicular tissue remain pervious to air, still the tension under which the air is retained will be increased, and the elasticity of the tissues will be lessened, inversely, according to the increase of the fluid. Finally, a pleurisy with effusion will alter the relations sustained by the thoracic viscera to the chest surface. If we are aware of the size of the areas occupied by these organs, we can readily detect them in any situation.

Diagnosis of Acute Pleurisy. *Inspection* is not always possible in private practice. The effects of the above changes are a notable lessening of respiratory movement, due not so much to the lesions as to the pain caused by the respiratory movements, which are not, therefore, attempted. There

is also a decrease in the movements of expansion and retraction of the affected side. It is not true, as often stated, that respiratory movement is abolished; on the contrary, there is under almost all circumstances an up and down movement of the ribs on the affected side, which betokens their elevation in common with the non-affected side; but expansion and retraction are subtracted.*

Increasing Effusion. The more fluid contained within the chest, the more distended will be the affected side; the interspaces will become filled out to a level with the ribs. There will be an increasing want of expansion and retraction, although, even when the chest is fullest, there is more or less up and down movement.

Inspection in Increasing Effusion. If the effusion is on the left side, we note that the heart is displaced to the right, proportionately to the amount of effusion; the apex may come to pulsate in the epigastrium, or entirely to the right of the sternal bone, even in the right axilla anywhere from the second to the sixth interspace, and the whole ventricular outline may be bounded by percussion. The pulsation to the right of the sternum is sometimes due to the shock of the entire ventricle against the ribs. On the other hand, similar displacements of the heart to the left side occur in right-sided effusions, only the displacement is not so great.†

Mensuration in Pleural Effusion. The tracing of the contour of the chest on paper will show a departure from the physiological shape. The accumulation of fluid gives a more rotund appearance to the chest walls, although the circum-

* *See* section on Modifications of Respiratory Rhythm, and contrast intercostal neuralgia with pleurisy.

† Displacements of apex-beat are a notable symptom of chronic pleurisy, of fibroid phthisis, and in the various diseases of the cardiac valves; conditions readily differentiated from pleural effusion. (*See* also section on Emphysema, for summary of epigastric pulsations.)

ference in inches may or may not be larger upon the side of the effusion, and is frequently less than the healthy side. This difference in size is abetted by the tendency of the chest walls to retract as the effusion diminishes.

The unaffected side will be enlarged to a greater or less extent, since it is practically in a state of supplemental emphysema, but the interspaces will not be bulged. This enlargement of the non-affected side is to be explained by the fact that the vicariously dilated lung tissue has not sufficient elasticity to oppose to the eccentric resiliency of the chest walls. It is wonderful to note how little dyspnœa is caused by the presence of large effusions in the chest. The rapidity with which the fluid accumulates in pleurisy with effusion, bears a direct relation to the amount of dyspnœa. So we may set down for a clinical fact, that complete filling of one pleural sac may occur gradually, without noticeable dyspnœa. On the other hand, the respiratory efforts on the unaffected side may be painfully exaggerated. In the initial stages of a pleurisy with effusion, there is often much dyspnœa, from the pain caused by efforts to secure complete respiratory action. The distress seems greater than in many cases of pneumonia.*

Exceptional Physical Signs in Pleural Effusion. In certain cases an actual in and out movement of the interspaces of the involved side may occur, synchronous with respiration. This may occur even if an effusion nearly fills one of the pleural cavities, but is especially noticeable in cases of lesser effusions and those of subacute or chronic type, particularly when they are undergoing absorption. There is frequently no obliteration of the intercostal spaces, and sometimes the depression of the intercostal tissues occurs only on inspiration. The explanation of these movements of the intercostal tissues may be as follows: The longer a fluid effusion persists, the more dense

* *See* Intercostal Neuralgia.

does it tend to become, and the area it occupies is diminished, just as a jelly when it forms occupies a slightly smaller area than the fluid out of which it was formed, and thus the intercostal spaces tend to sink inward in this class of cases. In addition, consider that during the time an effusion persists the chest walls are bulged beyond a physiological position; and from this point, owing to inherent resilience, they tend outward at each inspiration, but the lung elasticity is abrogated, to a varying degree, by the compression of the pulmonary tissues by the effusion. Thus it comes to pass that the state of the chest walls resembles their condition in moderate emphysema; consequently, the pulmonary intercostal tissues are depressed on inspiration, and tend to puff out or return to their pathological state during expiration, just as these tissues act in emphysema of the lungs.

Movements of the intercostal tissues during respiration may be due to the union of the pulmonary and costal pleura by adhesions. In these cases the retraction of the tissues would occur during expiration only, not during inspiration. The tissues may retract only on inspiration, in cases of pressure or obstruction on some portion of the bronchial tree, but this cause of pressure need only be kept in mind, since it is easily determined.*

Percussion in Pleurisy. In the very early stages of pleurisy the evidence by percussion may be negative, since the conditions of the pleura already described may not sufficiently separate the lung from the chest wall to render the sounds positive. As the process progresses it results in diminished pulmonary expansion and consequent increased pulmonary tension, causing the percussion note to become dull. When an effusion has collected the note becomes flat, differing in this respect from the dull percussion in cases of condensation of pulmonary tissue. In these the percussion derives some degree of

* *See* also Capillary Bronchitis.

dullness, because we develop vibration in the air within the bronchial tubes. The flatness on percussion in pleural processes is directly the sequence of the separation of the lungs from the chest wall, by dense non-resonant collection. Naturally, as the effusion ascends the chest, the percussion resonance tends to become flat in proportionate areas. The sense of resistance imparted to the finger used as a pleximeter is very marked. Especially are the flatness and sense of resistance pronounced at the lower portion of the chest, since the fluid naturally settles there. The line of flatness is commonly said to be capable of changing its level in partial effusions, with the changes of the posture of a patient, and this is so in all cases in which the fluid is not circumscribed by adhesions. These, however, may limit the effusion into an indefinite number of pouches, of smaller or larger capacity. In these cases, the level of fluid cannot change, nor can the line of flatness. In any event, however, the line of flatness is higher behind than in front.* One is more justified in resorting to exploratory paracentesis to confirm the evidences of pleural lesions by percussion, than after the employment of any of the other methods in physical diagnosis. The flat note, it is true, is developed over pleural thickening or solid tumors, but the diagnosis in these cases is not so often a source of confusion as between pleural effusions and consolidations.

The significance of *percussion above the layer of liquid* is best understood by a thorough appreciation of the conditions under which the air yet remaining in the lung is placed, and the condition of the pulmonary tissue. The resilience of the pulmonary substance is more or less diminished, consequently the air is restrained in a state of tension, varying with the size of the effusion. The resonance, upon principles already discussed, becomes high-pitched in the immediate vicinity of

* *See* section on Respiratory Percussion used as a method to define the upper margin of a pleural effusion.

the liquid; the quality may be dull, or some tone of tympany, owing to the compression of the lobes of the lung. Notwithstanding this, the transition from the clear pulmonary tone to the flat percussion over the area of effusion is markedly abrupt. The only source of confusion is when some process of phthisis or pneumonia complicates the effusion. The differential diagnosis will be subsequently described.*

The tympanitic quality of the percussion resonance above the effusion is best appreciated in front of the chest, near the superior portion. It may be so high-pitched as to become almost amphoric, and in children, or those with thin and flexible chest walls, the cracked-pot resonance can be developed. One caution must be observed in percussion of small effusions on the right side posteriorly, viz., not to mistake the line of hepatic dullness for the line of an effusion; in this case, change of posture often resolves the difficulty.

On the non-affected side, the pulmonary resonance is vesiculo-tympanitic.

Palpation in Pleurisy. This method affords an excellent means of diagnosis of pleurisies. The waves of sound are prevented from radiating to the surface by the separation of the pulmonary tissue from the chest wall, implied by this disease. This is quite the reverse of the finding in cases of consolidations of the lungs, in which the vocal fremitus is increased. The evidence of palpation is always valuable, whenever the tone of voice is sufficiently sonorous to develop any fremitus on the unaffected side. With these premises, the absence of vocal fremitus is a reliable sign of pleurisy, the more pronounced the effusions the more positive is the sign.

Auscultation in Pleurisy. In the primary stages of pleurisy, the principal changes in the respiratory murmur pertain to

* *See* Respiratory Percussion on this point.

rhythm; inspiration and expiration are shortened; the respiratory murmur is feeble, chiefly because of the pain caused by the respiratory act. Associated with the murmur are râles, of the dry or moist variety of friction. Enough has been said, in the chapter devoted to râles to guide in their separation from intra-pulmonary râles; sometimes we establish the diagnosis of pleurisy by considering the general symptomatology of this condition with the physical diagnosis, and thus distinguish between sounds often acoustically identical with intra-pulmonary râles.*

As the effusion rises, the respiratory murmur may be suppressed. Paradoxic as the expression seems, we hear silence all over the region of the effusion.

The Vocal Resonance in Pleurisy is not heard over the region occupied by the fluid. Above the level of fluid the respiratory murmur is exaggerated or broncho-vesicular, according to the degree of compression of the vesicular pulmonary substance. In many cases the respiration at the apex, on the affected side, anteriorly, may simulate the amphoric. To prevent mistakes from this source, note that the amphoric or cavernous sound is not so hollow as that which indicates a cavity. Further, on auscultation or percussion, consider the abrupt line below which respiration ceases, or percussion resonance becomes flat, a line so abrupt that the transition is often best appreciated by auscultation with the stethoscope. Consider also the associated symptomatology, which is very distinctive in both phthisis and pleurisy.

We may have a widely different result in auscultation of pleural effusions from that given above.

The respiratory murmur may be bronchial or tubular, almost identical with the breathing in phthisis or pneumonia. How shall we differentiate? Let us note that, in consoli-

* Deviations in respiratory rhythm for diagnosis of pleurodynia.

dation of the lungs, the bronchial or tubular breathing has a brazen ring, which carries with it the impression of superficialness of origin. On the other hand, the plane at which the bronchial breathing of pleural effusion is developed is manifestly deeper. The sound is more distant and muffled, giving the impression that the respiratory murmur is being carried on at a distance. This impression is more readily conveyed by illustration at a bedside, than can be stated in language, and it is more easily grasped in the clinical study, than might be fancied from the description.

The vocal resonance shares in the modification. It is not suppressed, but may be heard very loudly as bronchophony. At the same time, it is separated from the bronchophony indicative of consolidation, by the deeper plane in which it seems to be developed. Near the spinal column, posteriorly, the bronchial breathing and the bronchophony are very constant. This is, probably, because, in a majority of instances, this is the situation of the lung.

The class of cases in which the above physical signs are observed comprise, as a rule, those in which the effusion partially fills the chest ; the air which enters the lung above the fluid generates bronchial breathing, and the murmur is transmitted downward—over the area of effusion. In cases of sacculated effusion, the loculi being small, the conditions of condensation and tension brought about by the bands of lymph may favor conduction ; in these cases, the respiratory murmur is heard as a distant bronchial sound. Carefully going over the chest with a stethoscope, will oftentimes enable the observer to note areas over which the murmur is much more distant, muffled or absent, than others ; these spots so selected are often the areas of effusion. On the whole, whatever be the real reason for the persistence of bronchial breathing, the most useful distinguishing feature is the element of distance, or muffled quality of the

murmur. Now the persistence of bronchophony is explicable upon the same grounds. The element of distance, or the muffled quality of the sound, is the only element to enable us to make correct inferences. Its increase above the line of a pleural effusion is attributed by Dr. James (see *Edinburgh Journal*, Oct., 1880) not to condensation of the lung above the fluid, but to the waves of sound being transmitted through the bronchial tubes to the effusion and reflected upward, the ear appreciating the resonance physiologically diffused over the entire thoracic cavity. This increased resonance can be heard at any point of the chest above the effusion. Skoda, however, located it at the lower angle of the scapulæ, near the large bronchi, near the vertebræ, and above or below this line. Guttman declares the clear bronchial vocal resonance is only heard when the dense lung tissue touches the chest, usually between the vertebral column and the scapulæ.

Egophony can be described as a modified, or split bronchophony. By some it has been compared to the bleating of a goat. The sound is high pitched, with a nasal, ringing quality. It is usually heard near the angle of the scapulæ or in the axillary region. It is indicative of an effusion partially filling the pleural sac, and is heard at the upper margin of the effusion where there is a thin layer of fluid between the lung and the chest wall. It is a sound which has only a relative importance.

Physical Signs in Pleurisy During Absorption. We need not pause long to indicate the retrograde physical signs of diminishing effusion. For a long time after vocal resonance and fremitus have returned, and the respiration is again heard, it continues enfeebled, and its character is neither vesicular nor purely bronchial. The friction sounds may reappear as the roughened surfaces again come in contact; finally, the percussion very gradually becomes normal.

Frequently, more or less false membrane unite the two pleuræ, the intercostal spaces resume their normal shape, and

the chest is either restored to its natural size, or is left permanently somewhat contracted. The bronchial breathing near the vertebral column persists for a long time, since a lung that has been compressed unfolds but slowly. The usual general symptomatology, and physical signs of pleurisy, as contrasted with pneumonia, are appreciated by the following table, in the main abstracted from Dr. Da Costa's diagnosis :—

PLEURISY.	PNEUMONIA.
Sharp pain; not necessarily referred to the affected side.	Dull pain.
Friction sound; dry cough; impaired chest motion.	Crepitant râle; cough followed by expectoration.
In stage of effusion, obliteration of intercostal spaces; enlargement of side; displacement of several viscera. Exceptions frequent (see text); absence of expansion but persistence of up and down motion of ribs.	In stage of hepatization none of these signs are manifest.
In a large majority of cases, flatness, with absent, or distant and muffled bronchial breathing, and voice signs correspond.	Dullness with marked bronchial respiration, brazen, and near the ear, distinct thoracic voice, that is bronchophony.
Vocal fremitus abolished.	Vocal fremitus increased.
Decubitus is often in the affected side.	Decubitus not peculiar, sometimes on the sound side.
Sputa frothy, rarely any râles in the chest.	Sputa rusty colored, râles from accompanying bronchial inflammation common.
Febrile symptoms usually slight.	Febrile symptoms severe.
Temperature record irregular, not characteristic; rarely high.	Temperature record much more characteristic. Temperature rises rapidly, soon after onset; then is continuous, with marked evening exacerbations, from two to three degrees, and morning remission. Often reaches 105°. May show sudden elevations and striking falls in the whole course of the fever. Toward the end of disease generally rapid.
	High temperatures, with delirium, especially common in pneumonia of upper lobe.

Differential Diagnosis. In pleurisy, as in pneumonia, tympanitic distention of the abdomen may secure an elevation of the diaphragm, and thus modify the results of percussion and auscultation, by creating tympanitic percussion, and also by suppressing the respiratory murmur. These physical signs, taken together, are easily understood. On the left side a drink of water will often settle the question, since the ear, if applied, will hear the râle in the stomach. There is always the possibility of confounding the severe pain of neuralgia of intercostal nerves or muscles, with pleurisy. The chief point of similarity is the restriction of the respiratory movements. There are, however, no râles in pleurodynia; there is also pain, with tenderness on pressure, much aggravated by movements of the arm. The pain is of two kinds, either definitely located in the course of the affected nerves, or else the pain, if muscular, is fugitive and shifting, attacking often both sides of the chest. There is tenderness with pain in pleurodynia; in pleurisy there is pain, usually without tenderness. In pleurodynia there is no fever, which is a symptom of pleurisy.

Pleurodynia is a source of great suffering to phthisical patients, and in their case it is of considerable importance to discriminate from a therapeutic standpoint.

It is often very difficult to establish, positively, the diagnosis of pleuro-pneumonia. The physical signs of the two diseases, pleurisy and pneumonia, undergo modifications by the blending of lesions. The significant physical signs of pleurisy are the muffled respiratory murmur, becoming more distant in proportion to the increase of the fluid. There may be friction sounds in the early stages. On the whole, we are inclined to place great confidence in the absolute flatness of the percussion note over the level of fluid in these cases, since of all cases of pleural effusion, those connected with pneumonia are most apt to present us with a very distinct bronchial

breathing, often hardly to be differentiated from that indicating consolidation. Next to percussion, the vocal fremitus is most reliable in its results.* †

Hydrothorax.—This term is commonly applied to those effusions in which the accumulation occurs from causes other than inflammatory. In a word, the condition is one of dropsy, and is associated with diseases of the heart, kidneys, liver, or systemic states influencing the crasis of the blood. Important distinguishing points are that it is often associated with general dropsy. Hydrothorax is bilateral; pleurisy, even if bilateral (which is rare), can, perhaps, be antecedently located in one or the other side, or it can be traced to an altered blood crasis, the result of deficient assimilation or elimination. There is also an absence of frictions, or a history justifying the hypothesis of antecedent inflammation. Pericardial accumulation, enlargement of the heart or liver, intra-thoracic tumor, emphysema or pneumothorax, may be mistaken for pleurisy with effusion.

* *See* also Respiratory Percussion.

† Dr. Flint has reported a case of serous pleural effusion demonstrated by paracentesis, in the *American Journal of Medical Science*, April, 1882. It is remarkable, because the vocal resonance and fremitus were somewhat greater on the diseased than on the unaffected side. The vocal resonance was not bronchophony but had the characteristics of normal vocal resonance as distinguished from those of bronchophony. Dr. Flint reports the clinical fact without attempting explanation.

CHAPTER XII.

LESIONS AFFECTING THE PLEURAL CAVITIES. CHRONIC PLEURISY.

Chronic pleurisy properly includes the results of persistent inflammations, even the states of effusion already described. We shall restrict its meaning to the description of inflammatory thickenings of pleural membranes, and to the conditions under which the pleural cavity is divided into compartments by false membranes inclosing a considerable amount of fluid. In chronic pleurisy the process may indicate merely fibrous thickening of the pleura by filamentous or membranous adhesions; or the pleura may be enormously thickened, and, as subsequent contraction is inevitable, the pleural cavity may be obliterated. If there has been pus in the effusion, we may find a more or less complete cuirass enclosing the pleural cavity. The deposit is formed upon both the visceral and the parietal pleura, and is composed of calcareous plates of varying thickness. Sometimes pleural thickenings are associated with chronic interstitial pneumonia. Pleural adhesions are associated with most of the forms of phthisis. In the course of chronic suppurative pleurisy, lesions of the ribs, periostitis, necrosis, exostosis, etc., are quite common.

The diagnosis of encysted effusion has been already dwelt upon. In addition, local prominences are combined with physical signs of effusion, and a clinical history of pleurisy. In these cases, bronchial breathing may be very distinct, the vocal resonance very clear. Examine carefully the surface of the chest, for the areas over which these physical signs are most distant and muffled. Make use of the stethoscope, since the ear takes cognizance of the sounds developed over too wide an area. Percuss carefully, and it may be possible

to develop pulmonary resonance around sites of flatness. The pulmonary resonance is, in these cases, developed from portions of lung tissue, which may be drawn into corners of the chest by the adhesions, and completely surround a pouch of fluid. Bear in mind that these projections of lung may themselves be involved in a process of chronic inflammation, mostly of fibroid type. On the left side, it is possible to mistake enlargements of the heart for pleural effusion. The pulsation of an enlarged ventricle is peristaltic, and can be located as belonging to the heart by auscultation of the rhythm of the shock. If transmitted pulsation exists, the pulsation is not peristaltic, nor is it as expansile as in direct pulsation.* Aneurisms and tumors may also be sources of error. Analyses of such cases are among the intricate problems of diagnosis, and require an application of our conceptions of the principles underlying general as well as physical diagnosis.

The various degrees of thickening of the pleura produce important changes in the perimeter and contour of the affected side. The expansion of the lung, and its elasticity, are more or less modified; the eccentric resiliency of the chest-walls is overcome by the contractile power of the newly formed tissues. A solid union may be formed between the pleural walls, the ribs may be drawn together and approach one another, especially at the lower part of the chest, so that the diseased side becomes much smaller than the healthy side. The unphysiological contour is best displayed by tracings, the inferior part of the chest being most altered. Retractions at the base of the chest usually denote chronic

*Yet this rule has exceptions (*see Philadelphia Medical Times*, May 21st, 1881, p. 537). Dr. Henry made a few remarks upon a case of pulsating emphysema in which expansile pulsation was an important feature.

pleurisy. Reversely, retractions at the apex denote probable phthisis.

Percussion in Chronic Pleurisy. The pitch and quality of the note must vary, from dullness to flatness, in accordance with the amount of air permitted to enter the chest; with the thickness of the pleural membranes; or with the thickness or thinness of the external thoracic walls; and in accordance with the state of the abdominal viscera. It is in these cases most particularly that the tympanitic distention of the stomach or abdominal viscera may mislead.

Auscultation in Chronic Pleurisy. Precisely similar conditions influence the respiratory murmur. The rhythm is interrupted, the intensity is very feeble, or, if in the compressed and possibly fibroid lung the bronchial tubes are pervious to air, the respiratory murmur may be bronchial or even tubular, perhaps more or less muffled or distant in quality.

Vocal Resonance in Chronic Pleurisy is of variable assistance, and must be considered as corresponding to the respiratory murmur. If the voice is sonorous, the vocal resonance is well pronounced. It is more concentrated and louder than over normal areas, because the adhesions are favorable to the conduction of the voice, but the vocal resonance is more muffled and distant than it would be in a case of intrapulmonary lesion.

Palpation in Chronic Pleurisy. Vocal fremitus is absent, but sometimes, if the separation of chest walls from the lung is not very great, and there is a fair pulmonary expansion, some vocal fremitus can be felt.

CHAPTER XIII.

LESIONS AFFECTING THE PLEURAL CAVITIES. EMPYEMA. PNEUMOTHORAX.

Finally, as stated, pleurisy may terminate in empyema; but it may be purulent from the outset. Sometimes purulent pleurisy is the result of infection from purulent foci situated elsewhere. The pleural inflammation of pleuro-pneumonia is sometimes suppurative; the same is true of some pleural inflammations associated with phthisis. Collections of pus in the chest wall may follow wounds or fractures of the ribs; or peri-hepatic inflammations, or abscesses of the liver may perforate the pleura, and cause empyema. The quality of the pus is usually laudable, but in pyæmic empyema, or when there is communication with the external air, the pus may have a fetid odor.

Empyema may last months or years, terminating in evacuation of the pus externally, through an intercostal space, or it may be evacuated through a bronchus. More rarely, empyema passes through the diaphragm into the peritoneum or colon, or the pus empties into the mediastinum, thence along the vertebral column as far as the psoas muscle; or through the tissues forming the abdominal wall, and so outward. The termination of spontaneous external evacuation, through an intercostal space, may be preceded by periostitis, with subsequent caries or necrosis of one or more of the ribs, and it is preceded by an œdema of the intercostal tissues. The pleural tissue in long standing empyema may be converted into a complete so-called pyogenic membrane. The lung may occupy the positions already described in pleurisy with effu-

sion, or it may be bound down flatly against the diaphragm. The substance of the lung may be hepatized, or the compression may result in almost complete atrophy of the lung. The physical signs in general are much the same as in other varieties of pleural effusion.

Inspection in Empyema. Contractions or adhesions of the pleura may prevent as much displacement of the heart and diaphragm as in ordinary pleurisy. There is a very constantly increased circumference of the chest, and bulging of the intercostal spaces of the affected side. There is often a unilateral œdema of the corresponding side of the trunk. Dr. James, *Edinburgh Journal*, October, 1880, says: "In empyema these signs are due to the more acute pleural inflammation, producing at the same time a greater amount of paralysis, or possibly inflammatory alterations in the underlying intercostal tissues. Thus occurs greater intra-pleural tension through diminished absorption, resulting from an interference with the respiratory movements. The lymph circulation in the intercostal spaces is dependent on the movements of the intercostal muscles, just as the circulation in the diaphragm is dependent on its movements. In this way the lymphatic circulation is doubly disabled." Yet this is not invariable, since unilateral œdema is present in cases of hemorrhagic, and sometimes in simple pleural effusion. On the whole, however, if unilateral œdema of the chest walls is present, we may not expect a simple serous effusion.

The Vocal Resonance in Empyema is apt to be very distinct all over the chest surface, possibly because of the presence of adhesions and the thickened pleural tissues favoring conduction. Careful auscultation will show that at some points on the chest wall the vocal resonance is more muffled than elsewhere. These cases are often those of sacculated effusion,

and the points of muffled vocal resonance offer most favorable spots for paracentesis.*

Local surface thermometry may become an available means of establishing the diagnosis of empyema, showing an increase of temperature upon the affected side. Clinical observation will, perhaps, some day, differentiate the diurnal waves of temperature in cases of internal abscesses. This has been done in abscess of the liver, in which case the remittent type, and a comparatively low range of temperature, 100° to 102½° F., is the rule. But in empyema we have a large abscess, which, in common with internal collections of pus, gives expression to a group of manifestations, embracing marked fluctuations in temperature, sweatings, chills, in fact, a hectic fever. It is the duty of the physician, with the above ascertained facts on the one hand and the physical signs of pleural effusion on the other, to solve the problem, by the exploratory puncture with aspirator or hypodermic needle. Strange as it may seem, large collections of pus may persist in the chest without much rise in temperature. The cases in which this observation has been made would seem to be those cases of chronic pulmonary and pleural disease in which the pleura are thickened and indurated. Perhaps, in this condition the pleural thickening opposes a sufficient barrier to the development of renewed inflammation from fresh pulmonary catarrhs. The symptoms are so latent that the pus may remain hermetically encapsuled for months or years.

Hæmothorax proceeds, usually, from the rupture of some

* Dr. Bacelli maintains that if the liquid be serous the whispered voice is well transmitted through the liquid, and is pectoriloquous in character. On the other hand, the voice is not well conducted if the liquid be purulent. Guineau de Mussy sustains the correctness of these observations.

Dr. Douglas Powell has published a series of cases, some of which sustained, and others conflicted with the observations of Bacelli.

of the newly-formed blood vessels, in an attack of pleurisy, unless it is associated with new formations in the pleura. The operation of paracentesis often first reveals it.

Pneumothorax. This condition represents the accumulation of air in the pleural cavity. As a rule, it is consequent upon some prior disease of the lung. It may originate when there is but moderate pulmonary disease, by perforation of the visceral pleura covering a patch of superficial consolidation, or by the ulceration of the walls of a small superficial cavity. More commonly, perforation of the visceral pleura happens in advanced phthisis. "Da Costa gives a rare mode of origin by the rupture of a distended air vesicle." It is also true that the air may enter the pleural sac by the perforation of the pleura from without, in cases of wounds, or during the operation of paracentesis. The transit of air from the lung into the pleura is attended usually with great pain, together with a sudden development of intense dyspnœa. The condition occurs, as a rule, during a paroxysm of coughing. Death may speedily follow, but if not, the accident is usually followed by the development of pleurisy with effusion, which may soon become purulent. This condition is known as hydro-pneumothorax, which embraces the majority of cases, simple pneumothorax being comparatively rare. The disposition of the lung when pneumothorax occurs is similar to its disposition in pleural effusions. If, however, there are many adhesions prior to the formation of the pneumothorax, the collections of air may be loculated or pouch-like, surrounded by the pulmonary tissue. The fistula connecting the lungs with the pleural cavity may be closed by inflammation after a variable period, or the orifice may remain patulous.

Percussion in Pneumothorax. Naturally the sound must be tympanitic or hollow. The pitch is lower than the tympany

of most cavities; it is lower than the pitch of the stomach tympany. The reason is that the pleural cavity is large, its walls are tense, consequently the vibrations of the pulses of air are rapid, and the quality of the sound is tympanitic.

Auscultatory Percussion in Pneumothorax. If there is a patulous intra- or extra-pulmonary fistula, auscultation practiced while percussion is made by means of two gold pieces, a beautifully clear, ringing, metallic sound will be heard. At times, although the pneumothorax may be large, the lung is bound down by adhesions, and the orifice of communication between the pleura and the lung may be closed; the percussion becomes so high pitched, that its quality may be imperfectly developed. This fact, and the possible associated conditions of thickened pleura, or thick layers of external tissues, render the sound at times a little dull. Tympanitic dullness is the term best adapted for these cases of pneumothorax.

Auscultation in Pneumothorax. Entire suppression of respiratory murmur is the rule in pneumothorax. It is more absolute than in the similar separation of the lung from the chest wall which occurs in pleurisy, owing to the great compression exerted by the air upon the lung. In some cases, the conditions surrounding the perforation in the visceral pleura may keep the fistula more patulous than in others. When it is patulous, the air rushing into the artificial cavity formed by the separated pleural surfaces imparts to the respiration an amphoric, cavernous tone. If drops of fluid escape from the lung, and fall into the cavity, or if bubbles of fluid are generated on the surface of the pleural effusion, the sounds are often echoed to the ear with a metallic ring, resembling the tinkle of a silver bell.* The presence of this sound signifies the patulous state of the pulmonary fistula;

* A large cavity may yield this tinkle, or some liquid in a large bronchial tube.

its absence does not necessarily mean that the opening is closed permanently by inflammation, since the closure may have been effected by other causes, such as partial inflation of the lung, position, and the like. The compressed lung may be detected by a limited area of bronchial breathing, usually near the spinal column. When, in exceptional cases, there are many pleural adhesions prior to the formation of the pneumothorax, some form of bronchial breathing will be heard in different areas in the chest. A diagnosis in these cases is very difficult, and is only effected by summing up all the physical signs and general symptoms.

Succussion is an audible splashing sound which can be developed in pneumothorax by abruptly shaking the patient. The succussion can be detected as a fremitus.

Inspection in Pneumothorax. The ingress and egress of the air into the pleural cavity widens the chest, and the intercostal spaces also become effaced. In other respects the remarks on the diagnosis of unilateral pleurisy by inspection and mensuration, are apropos. The chest is more motionless than in simple pleural effusion, though some up and down movement of the ribs may occur. Displacements of the intrathoracic viscera occur, as in pleurisies with simple effusion.

Vocal resonance and fremitus in pneumothorax are negative.

Differential Diagnosis in Pneumothorax. The recognition of the fluid in the base of the chest is dependent on the ordinary signs present in uncomplicated pleural effusions. Bilateral emphysema is frequent. Pneumothorax is almost always unilateral. Cases of bilateral pneumothorax have been cited, but they are so unusual as to be among the curiosities of medical literature. From unilateral emphysema remark: in pneumothorax, the suppressed respiratory murmur, and the existence of the metallic tinkle; succussion is also available.

The displacement of the viscera differs from emphysema. The percussion pitch is lower and the tone more hollow than in emphysema. The results of auscultatory percussion are very valuable. Finally, the antecedents of emphysema differ widely from pneumothorax, beside which the latter is suddenly developed. A distended stomach may become a source of error. If some water is sipped, the tinkle following its entrance into the stomach is very audible.

Large cavities at the base might mislead, particularly if the pneumothorax is encysted. In diagnosis, if we nucleate the facts gathered from the clinical histories, we find the evident signs of wasting bilateral disease, and the respiration is audible throughout a large area of super-imposed lung. In pneumothorax the pitch of the tympany is lower than in cavities.

CHAPTER XIV.

MEDIASTINAL DISEASE.

The limits of this volume restrict the observations upon malignant pulmonary disease principally to a description of the physical signs contingent upon mediastinal enlargements. Malignant disease occurs in two forms, the primary and secondary; the history, both personal and hereditary, should be carefully analyzed. In a case of apparent primary cancer of the lung, the diagnosis was finally decided in favor of malignant disease, because fifteen years before the patient had undergone an amputation of the leg for "some tumor." In malignant disease, pleural effusion is a frequent concomitant. It may arise through pressure upon the azygos or hemi-azygos veins, thus preventing a free return of blood from the pleural veins, with resultant hydrothorax; or the effusion may be the result of inflammatory process, the sequel to malignant disease, developed secondarily in the pleura. In this case the fluid is usually turbid, highly albuminous, with a large proportion of coagulable fibrine. The presence of a nodule or nodules of malignant nature in the lungs, when large enough to be appreciated by physical diagnosis, yield a very dull or flat note on percussion. The respiratory murmur is some phase of bronchial breathing, or if a bronchus is pressed on, may be feeble or suppressed over the affected side. Over the bronchial tree, behind the third dorsal vertebra, the breathing is feeble or whistling if the primary bronchi are compressed. If bronchitis exists the sputa may be purulent; if the new formation undergoes softening, with formation of cavity, the expectoration may be bloody, resembling prune juice or black currant jelly. The cells constituting the growth, with portions of pulmonary structure, may be found on microscopic examina-

tion of the sputa. Primary cancer of the lungs, or that propagated from the mediastinum, is usually unilateral; if secondary to disease elsewhere, it may be bilateral. Inspection may show retraction or expansion of the chest, necessarily the latter, if pleural effusion exist.

In cases of pulmonary cancer, if we examine the supraclavicular regions by palpation, we can often discover masses of painless, movable glandular enlargements. The nodules in the lungs vary, from the size of a chestnut to a lemon, or diffused cancerous infiltration may be disposed throughout the various lobes. Cough, pain, or dyspnœa, may develop the impression that phthisis, especially the fibroid variety, coexists. Even pyrexia may become a symptom, with night sweats confined to the affected side (Walsh). Still the elevation of temperature is less than in phthisis. Phthisis is a rare coincidence of the history of malignant disease, and occurs mostly at an earlier period of life. Walsh asserts a mean duration of pulmonary cancer to be 13.2 months, a minimum of 3.5 months, the maximum at 27 months; but he acknowledges that this is based on a small contingent of cases. Bronchitis, with atrophic emphysema, simple pleural effusion, chronic pleurisy with retraction, fibroid phthisis, are the diseases with which it is most likely to confuse a case of cancerous disease. The characteristic history of these diseases, elsewhere detailed, the cancerous hue of the skin, and the history of the case, are helpful evidences on the side of malignant tumor.

Mediastinal tumors include enlargements of the bronchial glands, in the malignant process, in syphilis, in scrofulous persons, in some cases of pulmonary catarrhs, or phthisis, and in some cases of pertussis, or rachitis. Prominently in this connection are the physical signs demonstrated by M. Guineau de Mussy by percussion over the spinous processes of the cervical vertebræ, in the course of the trachea. Follow-

ing this line in the healthy subject, a distinct tubular sound is elicited by percussion down to the point of bifurcation of the trachea at the level of the fourth dorsal vertebra. Opposite the fifth and downward we get the lower-pitched pulmonary resonance. When the tracheal and bronchial glands are enlarged, the tubular sound over the upper dorsal vertebræ is replaced by dullness, which may contrast sharply above with the tracheal, and below with the vesicular resonance.*

The respiratory murmur will be feeble in volume and limited to inspiration, especially over the interscapular region. Over one or the other bronchus, the respiratory murmur may be more high pitched than in health, and slightly exaggerated on one side, or at the base of the chest. The rhythm is often jerky and paroxysmal; the paroxysms are more or less constant, but are liable at times to increase. The general symptoms of enlargement of the bronchial glands have been alluded to in the chapter on syphilis of the lungs. They include pain in the scapular region, often lessened by change of posture, with a sense of fullness and substernal tenderness. There is often a subjective sense of inability to thoroughly inflate the lungs, and attacks of dyspnœa resembling the asthmatic type sometimes occur. Aneurisms occur as mediastinal tumors. Any form of mediastinal enlargement is capable of exciting serious mischief in the lungs—such as atelectasis, emphysema, and certain forms of pneumonia.

* A contribution to the History of Influenza, by Drs. White and Guiteras, Phila. *Med. Times*, April 10, 1880. It is also associated with some cases of aneurism. *See* Path. Trans., vol. 17, London.

CHAPTER XV.

RESPIRATORY PERCUSSION.

Respiratory percussion has already been described, as it can be practiced on the normal chest. It remains for us to indicate its value in the examinations made in disease. Manifestly the method depends on the pervious state of the air passages and the vesicles, and upon the principles of physical diagnosis already laid down. Each observer will perhaps extend the scope of the method for himself. The writer has practiced the percussion in the following conditions, and unites with Dr. DaCosta in his conclusions: "In marked emphysema the excessively clear vesiculo-tympanitic note is unchanged by percussion during the act of breathing; when the emphysema is not so great, it is but slightly changed. But if emphysema be present at all, except to a trifling degree, the sound is not much altered." Since emphysema, with attendant enlarged and dilated right ventricle, is one of the most serious complications of asthma and of chronic bronchitis, how valuable it becomes to add this corroborative test. In pleurisy, full inflation will strikingly bring out the abrupt contrast between the pulmonary resonance above and the flat note below. When pleurisy co-exists with pneumonia, and there is blowing breathing at the back of the lung, this method is very helpful. If there be pneumonic consolidation at the lower part of the chest, the flat note remains unchanged; and so will the dullness of the upper part be unchanged by forced respiration. But if the tubular breathing be simply from compression or condensation of the lung, and not from hepatization, decided clearness takes the place of the dullness."

"At the base of the chest, if there be chronic pneumonia,

the line of dullness changes in part, and if there is no sharp line of contrast between dullness and resonance developed on full breathing, it is consolidated lung. When, by respiratory percussion, the dullness at its uppermost limit becomes sharply defined, while it is unaltered below, it is an effusion." "In bronchitis the percussion resonance is, practically speaking, unaffected. Yet where extremely abundant secretions exist, and obscure breath sounds, the clearness of note may become impaired, and we are in doubt as to the state of the pulmonary textures. Respiratory percussion removes the doubt; the chest struck while in a full respiration returns a sound exactly corresponding to the sound we should obtain in health. If, however, there be an extension to the finer structures, and beginning consolidation, the note does not become fuller and more resonant, and the difference between the damaged point and the surrounding parts, or corresponding portions of the other side, is manifest. If, however, the lung be merely collapsed, respiratory percussion gives an almost normal sound, unless the collapse be extensive, and the power of expanding the lung be lost, or inflammation beset the collapsed lobules." "In acute lobar pneumonia, as resolution begins, the note heard on respiratory percussion is more resonant, more pulmonary. This change may show itself in advance of the râles redux."

"In heart disease there is often congestion so pronounced as to lead to apprehensions of consolidations; these are removed when forced inspiration clears up the suspected points."

"There is a single point linked with phthisis, which is useful in connection with what has been said in connection with feeble breathing, in cases of phthisis. When in a case of phthisis we find that the dullness on percussion is no longer modified by fixed inspiration, we have a certain test of the malady having progressed." "In pneumothorax, if inspiration

does not change the percussion note, the opening is closed. If the tympanitic or amphoric note is changed essentially, we may infer that the air still rushes from the lung into the artificial cavity in the pleura."

"In regard to the cavities, the pitch of the sound is raised so much that the quality sometimes appears dull, or that intermediate type, tympanitic dullness, may prevail." *

This method of diagnosis is especially useful in children, as a means of differentiating between capillary bronchitis and catarrhal pneumonia; the best opportunity is when the child gasps in the act of crying.

**See American Journal of Medical Sciences* for July, 1875.

PHYSICAL DIAGNOSIS.

PART II.

DISEASES OF THE HEART AND PERICARDIUM.

CHAPTER I.

PROCESSES WHICH DEVELOP CARDIAC AND ARTERIAL DISEASES.

The only successful method of diagnosis of cardiac disease, and the most successful plan for its therapeusis, consists mainly in taking a broad view of the patient's general condition; in grasping the cause by its effects, as well as by the local physical signs. The antecedents of cardiac lesions are important auxiliary aids to diagnosis. The causes of valvular disease or defect are:—

"1st Malformation of valves, arterial and auriculo-ventricular.

"2d. Injuries of valves, arterial and auriculo-ventricular; immediate and gradual.

"3d. Alterations in capacity of orifices and cavities.

"4th. Inflammatory affections, chiefly rheumatic, acute and chronic, are, however, the bases of the majority of cases" (Peacock). In a minority of instances, endocarditis is secondary to pleurisy, pneumonia, pericarditis or Bright's diseases.*

* "Taking 77 cases of cardiac disease in which a precise early history could be obtained, 34 occurred in those who had suffered one or more attacks of undoubted rheumatic fever; in 13, rheumatic pains, not sufficient to keep the patient in the house, had been noted. In 15 there was no

The cardiac substance is subject to hypertrophic and degenerative processes.

Hypertrophy occurs as a sequence of valvular lesions; of functional palpitation connected with over exercise, or abnormal innervation, sometimes induced by excessive use of tobacco, tea or coffee; of the puerperal state, and of chronic forms of Bright's diseases, or aortic atheroma. The connection between cardiac and renal lesion is especially important. When structural disease affects the kidney, elimination of the urinary solids is imperfect, and their retention results in vaso-motor irritation, with spasm of the walls of the arterioles. The passage of the blood through the arterioles is opposed, demand is made on the heart for increased propulsive power, and hypertrophy, chiefly limited to the left ventricle, ensues. If, however, renal disease develops in a patient already reduced in strength, with impoverished blood tissue, hypertrophy may not occur. In the more rapid examples of renal disease the increase of vaso-motor tonus is not sufficiently persistent to result in hypertrophy. In apposition, renal disease occurs as a sequence to cardiac disease, because the kidneys, like the other viscera, suffer venous repletion, and if this be long continued a form of mingled tubular degeneration and intra-tubular growth may result in the so-called cardiac kidney. The dropsy of secondary renal lesion begins in the feet instead of the face, the white skin and anæmia of renal dropsy is absent, instead of which we often observe evidences of venous congestion. Albuminuria and hyaline tube casts can be found on examination of the urine, but they disappear with the venous congestion.

history of any rheumatic affection whatever; if any symptoms were mentioned they were merely those of indigestion. Rheumatic gout had been the portion of 2 patients, scarlet fever of 3, and typhoid or low fever of 4. In 6 cases the evidence pointed to the conclusion that the disease was congenital" (Sansom).

Cardiac Degeneration concludes the history of hypertrophy consequent upon valvular lesions, or chronic Bright's diseases, because these processes induce degenerative changes in the aorta which lessen the activity of the circulation, or develop disease of the coronary arteries. Cardiac degeneration is also a part of the atrophic process inseparable from advanced age, but is associated with chronic and wasting forms of disease, and especially with the latter stages of renal and pulmonary disease, and in chronic alcoholism.

Dilatation of the heart is developed by over-exertion in those of feeble nutrition, and may ensue in both the extremes of life. It is frequently associated with emphysema and chronic forms of bronchitis. Dilatation is often combined with hypertrophy or with valvular lesions; forming the so-called eccentric or dilated hypertrophy.

Arterial Lesions. Arterial changes are usually the result of chronic inflammation, and terminate in atheroma. The syphilitic process, chronic alcoholism and the chronic forms of renal disease, are direct causes of arterial lesions. Increased arterial tension as it occurs in hypertrophy of the heart and the gouty state, tend to develop the same result. Atheroma may occur independently of the inflammatory process. In these cases the process of fatty degeneration is combined with calcareous deposition, and is very common in the latter periods of life.

CHAPTER II.

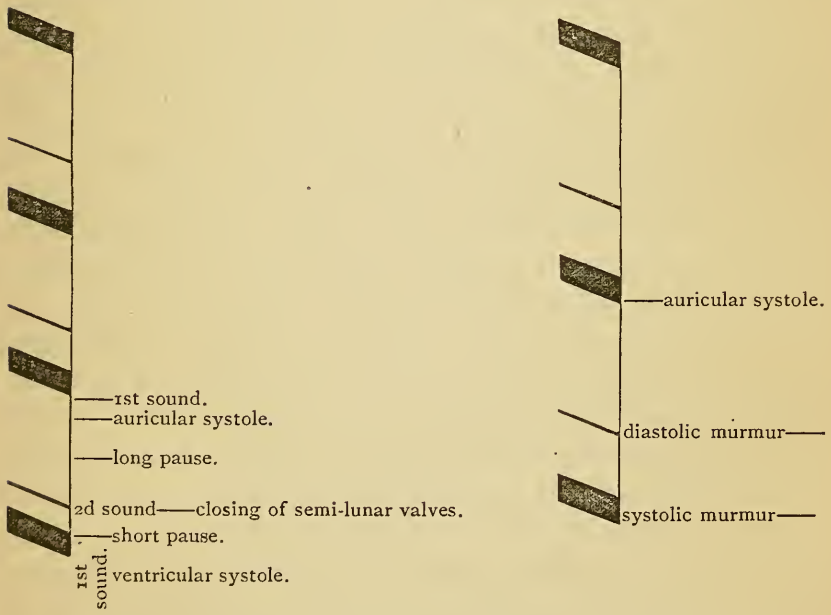
AUSCULTATION OF PHYSIOLOGICAL SOUNDS. AUSCULTATION OF MURMURS. THRILLS.

Physical diagnosis of the heart employs the senses of sight, touch and hearing. As auscultation is one of the most valuable methods of investigation, its application to the study of cardiac valvular disease will be primarily considered. It is necessary to become familiar with the use of the terms sounds and murmurs.

Auscultation of Physiological Sounds. During the normal revolution of the heart, audible phenomena occur, which we describe by the term cardiac sounds. The sounds of the heart are represented to the diagnostician by the terms first and second sounds. Several elements are combined to produce the first sound : it is partly the result of closure of the valves of the mitral and tricuspid orifices ; partly the whirl of the blood within the cavities of the ventricles ; partly the sound produced by the contraction of the muscular walls of the ventricles. This first sound, called systolic, represents the period during which the blood is projected from the left ventricle into the aorta, and from the right ventricle into the pulmonary artery. The interval between the systolic or first sound, and the so-called second or diastolic sound, comprises the short pause. The diastole, or long pause, includes the time during which the blood is flowing through the pulmonary artery and the lungs, via the pulmonary vein, into the left auricle, thence into the left ventricle again ; also the time occupied by the transit of the blood through the systemic circulation back to the right auricle and right ventricle. The diastole is a period marked at its commencement by the closure of the aortic semi-lunar valves and the like closure of the valves of the

pulmonary artery. This closure is effected by the systole or recoil of the aorta and pulmonary artery after their distention, which follows the ventricular contraction. The click connected with the closure of these semi-lunar valves is the accredited cause of the second sound. The diastolic interval, or long pause, following the closure of the semi-lunar valves is terminated by the contraction of the two auricles, by which they are thoroughly emptied of blood. The sound produced by the auricular systole is inaudible under physiological conditions, but the fact that an auricular systole occurs is important to a clear estimation of the murmurs developed by mitral obstruction.

The quality of the first and second sounds varies. The first sound is long, low-pitched and muffled; the second sound is high-pitched, sharp and clacking.



In disease both of these sounds become radically altered. If the first sound loses its muscular element, as in cases of

fatty degeneration of the heart, it may become less muffled, or even as valvular and clacking as the second sound, or the sound may be entirely lost. The second sound may become very markedly intensified, or accentuated, when the arterial tension is increased, or the sound may become feeble when the arterial tension is lowered. It is necessary, therefore, to possess more positive means of deciding which is first or second sound than the character of the sounds themselves, so we must ascertain the localities at which the sounds are best heard. It is an axiom, that both sounds and murmurs are most distinctly heard at that portion of the chest surface at which the cavity in which the sound or murmur is generated approaches most closely the chest surface. The left ventricle is most superficial in the region of the apex beat, and the aorta at the junction of the second and third ribs with the sternum. The right ventricle is most superficial near the ensiform cartilage; the pulmonary artery crosses in front of the aorta, and is most superficial near the junctions of the second and third ribs with the sternum.

We commence our study of a sound at the base, because we know that the valves are clustered at that point; we then proceed either downward to the apex, or upward to the second right costal cartilages. The first sound, therefore, is heard most distinctly at the apex, the second sound is most distinct at the aortic cartilage, although both sounds can be heard at any point of the præcordia. Practically speaking, we estimate the first and second sound as though they were produced only on the left side of the heart, but we can also hear distinctly the first sound over the xiphoid cartilage, at which place the right ventricle is most superficial, and the second sound can be also heard over the second left costal cartilage, where the pulmonary artery is most superficial. With a knowledge of the method of the production of a cardiac sound, and its distribution, we may

summarize the tests by which we differentiate the systolic and diastolic sounds.

1st Sound. The quality is muffled, long, low-pitched, phonetically represented by the word lupp. It is best heard at the apex beat. It is synchronous, not only with the apex beat, but also with the carotid pulse. It immediately precedes the radial pulse by a hardly noticeable interval.

2d Sound. The quality is clear, clacking and high-pitched, phonetically represented by the word dupp. It succeeds the apex beat and radial pulse. It is best heard at the second right costal cartilage, commonly called the aortic cartilage.

Auscultation of Murmurs. Heart disease, and especially valvular lesion, is most frequent on the left side. Diseases of the right side are usually secondary. Omitting all pathological details, the statement is true that the results of inflammation can alter each valve, so that its orifice, on the one hand, is roughened, contracted or stenosed. On the other hand, the valve may be nearly destroyed or thickened, and fringed with vegetations; or adhesions of the columnæ carneæ may bind down one or more leaflets, resulting in functional incompetency of the valve. In the mitral or tricuspid valves a clot may become so entangled in the columnæ carneæ, or chorda tendineæ, that insufficiency, or a murmur simulating insufficiency, may be produced. (*See* Thrombosis.) The vegetations and other products of inflammation upon the valves may undergo calcareous degeneration; but the semilunar valves may become diseased secondarily to atheroma of the aorta induced by arterial overstrain, or the degeneration consequent on advancing years.

The pathological sound induced by lesions of the orifices or modifications in the blood tissue are called murmurs. We have commonly five murmurs represented for our study:

three of insufficiency, with regurgitation; two of obstruction, or stenosis.

Valvular diseases of the right heart:

- Tricuspid valves
  - Obstruction or roughening very rare. Time pre-systolic.
  - Regurgitation.
    - Time, systolic.
    - Nearly always secondary.
- Pulmonary artery semi-lunar valves
  - Obstruction
  - Regurgitation

  Almost unknown unless congenital.

Valvular diseases of the left heart:

- Mitral valves
  - Roughening or obstruction — TIME.
    - Post-diastolic, pre-systolic.
    - Auriculo-systolic—occurs at end of diastolic period.
    - Mitral direct.
  - Regurgitation — TIME.
    - Systolic.
- Aortic semi-lunar valves.
  - Roughening or obstruction — TIME.
    - Systolic.
  - Regurgitation — TIME.
    - Diastolic at commencement of diastolic period.

The quality and pitch of murmurs are not indicative of the extent of the lesion. A most insignificant lesion may be manifested by a loud murmur, while a most serious lesion may give rise to a very insignificant murmur. The best method of estimating the gravity of murmurs indicative of valvular lesion, is by noticing the degree of change in the cavity in which the murmur is generated; and by estimating the degree of diminution of cardiac propulsive power, as manifested by a failing circulation.

Aortic murmurs are more frequent in persons of middle age and advanced life than in the young. Direct injury to the valves, resulting from laborious avocations, also prolonged high arterial tension, or atheroma, are more often the antecedents of the disease than rheumatism. This is especially true of aortic regurgitation. Mitral disease is more directly the most serious complication of acute rheumatism,

or the rheumatic diathesis. The rule is, however, not unvarying. Young persons suffer from aortic disease, particularly aortic obstruction, and old persons frequently present a long-standing mitral lesion.

Another point of interest relates to the pitch of murmurs. Mitral murmurs, especially if recent, are usually low-pitched, blowing sounds; aortic murmurs are usually high-pitched, cooing or grating sounds. The character of the pathological deposit bears a direct relation with the pitch of the murmur. If the auriculo-ventricular or semi-lunar valves become calcareous, or spiculæ project into the circulation, the murmur may become roughened and ringing.

A murmur may replace entirely the normal sound, or some portion of the normal sound may remain. In the latter case, the destruction of a valve is not so complete. Degenerations of the heart and weakness of cardiac muscular power can sometimes abolish a murmur previously distinct, the murmur reappearing if the cardiac power increases under treatment. The mitral murmurs resulting from acute rheumatic endocarditis may vanish in time, as the swelling subsides, and the edges of the valve again approximate normally. More commonly, the lesion increases in direct relation with the lapse of time and the tension maintained within the ventricle. If care is taken to conserve a minimum degree of intraventricular tension, by constant supervision (especially of the habits of life), the rapidity of the progress of the lesion is much reduced. Care must also be observed to prevent a repetition of attacks of acute rheumatism, each of which tends to aggravate the mischief. *All cardiac murmurs, like the cardiac sounds, can be heard at the base, and follow the law which prescribes their location of maximum intensity to that situation of the chest at which the cavity in which they are produced approaches most directly the surface of the chest. They are subject to another general law, viz., that they are propa-*

gated in the direction of the blood current by which they are developed.

Murmurs may obscure the normal sounds, but sometimes more or less of the latter may persist; this especially occurs in diseases of the aortic valves, and in such cases one may infer a degree of valvular competency.

A thrill is a purring tremor, or vibration, distinguished by palpation in the præcordial region. Thrills may be developed in the præcordial sac, and are then known as pericardial frictions. Thrills may also occur within the heart or great blood vessels. Endocardial thrills are coincident with the systolic or diastolic periods. They are developed by the whirling of the blood stream over roughnesses on degenerated valves, or at contracted valvular orifices, or, again, by roughnesses in the walls of the great vessels. A thrill is, therefore, developed in the same manner as a murmur, but only a small proportion of thrills are sufficiently violent to be sensible to palpation, so that very few audible murmurs are associated with thrills. They follow the same laws of location of maximum intensity as the audible murmurs. Both murmurs and thrills are frequently present in mitral obstruction. Thrills connected with mitral regurgitation are far from common. At the base of the sternum thrills may proceed from the aorta or the right ventricle. Both systolic and diastolic thrills can be recognized over the aortic region; their presence (excluding pericardial friction) indicates atheroma, aortic dilatation or saccular aneurism. The violence of the thrill, as of a murmur, can be increased by exertion. Thrills detected in the veins of the neck indicate a narrowed venous calibre, occasionally, from pressure.

CHAPTER III.

MITRAL REGURGITATION. MITRAL OBSTRUCTION. TRICUSPID REGURGITATION.

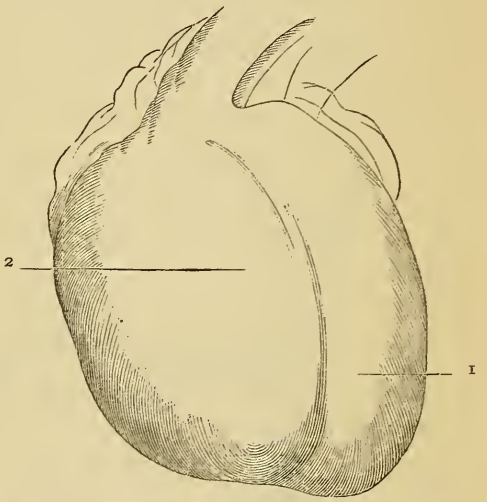
Mitral Regurgitation. *Auscultation.* This murmur is usually the result of rheumatic endocarditis. The valves are so altered that they cannot completely close the auriculo-ventricular orifice. When the process of inflammation affects the mitral valve, the recoil of blood within the ventricle during the systole impinges violently upon it, and the mischief is thus increased. To differentiate a mitral murmur we proceed as follows: Having heard a murmur at the base of the lung, auscult the aortic cartilage and then the apex. If the murmur be generated by regurgitation through the mitral valve, the murmur will be loudest at the apex. The next step will be to time the murmur, which can be done by noting that it is synchronous with the apex beat, or the carotid pulse; in other words, we time it as we would the first sound. Frequently, in initial lesions of the mitral valve in acute rheumatism, the murmur will not be transmitted outside the apex; but if there is much regurgitation, the murmur will be transmitted in the direction of the blood current, *e. g.*, back into the auricle, through the pulmonary vein into the lungs. The murmur can be traced to two sites on the chest, one the axilla (since the tissues covering the chest wall are thinnest at this point), and the other at the angle of the left scapula and the vertebral column. In the axilla always listen above the line of the apex beat; a recent murmur can often be heard at this point which may not be transmitted as far as the scapula. Sometimes the amount of swelling is only sufficient to thicken the first sound, or develop a slight murmur, heard at the apex and for a short distance outside; in this

case the amount of regurgitation is slight, and if the lesion occur in a case of acute rheumatism, the murmur may disappear after a time. A moderate lesion, if associated with much calcareous degeneration, may give origin to a murmur which may be transmitted to a great distance. Sometimes, when there is considerable regurgitation, mitral murmurs may be transmitted to the angle of the right scapula or right axilla. The murmur of mitral regurgitation may also be heard at the ensiform or the aortic cartilages, or over the auricle, but is evidently not so pronounced as in the line of the blood current, by which the murmur is developed. The pulmonary artery second sound is often accentuated, and the aortic second sound weakened.

Exceptions. Mitral regurgitation may exist in cases of extreme dilatation or degeneration of the heart muscle, and yet so little blood may regurgitate through the mitral valve at each systole, that a murmur is not developed. In these cases a murmur may return as the heart systole improves under rest and treatment. Sometimes a murmur may be heard in the erect but not in the recumbent posture, because of the increased action of the heart when the patient is standing or walking. In all these cases evidences of venous congestion prevail.

Effects of Mitral Regurgitation. The diagnosis of a mitral regurgitation is incomplete unless the study is interlaced with a consideration of the local and general consequences of valvular cardiac disease, and the gravity of the lesion must be deduced from the effects upon the cardiac substance and from the signs of falling circulation in the general system. The local effects of valvular disease find their primary expression upon that cavity of the four which is most directly affected by the abnormal circulation; afterward the adjacent cardiac cavities become more or less involved. The first demand of mitral insufficiency is an increase of propulsive power in the

ventricular systole. Hypertrophy is the response, but in most cases hypertrophy is inadequate to remedy the lesion permanently, or even for any considerable period. This arises, partly, from the fact that hypertrophy tends to force the blood through the abnormally patulous orifice, at the same time that it increases the force of the current into the aorta. The largest amount of blood naturally passes into the aorta, since the aortic is the larger orifice—and for a time the hypertrophy is compensatory. But all the while the increased power of the systole tends to increase, little by little, the regurgitation and widen the abnormal valvular orifice. It soon transpires that the aorta is inadequately filled—a condition fruitful of evil, because the coronary arteries are imperfectly filled by the imperfect aortic systole, and dilatation with fatty degeneration ensues. How rapidly, will depend on the degree of mitral lesion. The left auricle, in cases of long standing lesion, becomes hypertrophied and dilated; next ensues repletion of the pulmonary vein, with subsequent congestion of the lungs, with hypertrophy, and dilatation of the right ventricle. Enlargement of the right auricle occurs in its turn, with a more or less pronounced repletion of the entire venous system throughout the body.



CARDIAC LESIONS IN MITRAL REGURGITATIONS.

1. Moderate Enlargement of Left Ventricle. 2. Nearly Normal Right Ventricle. (Drawn from Specimen.)

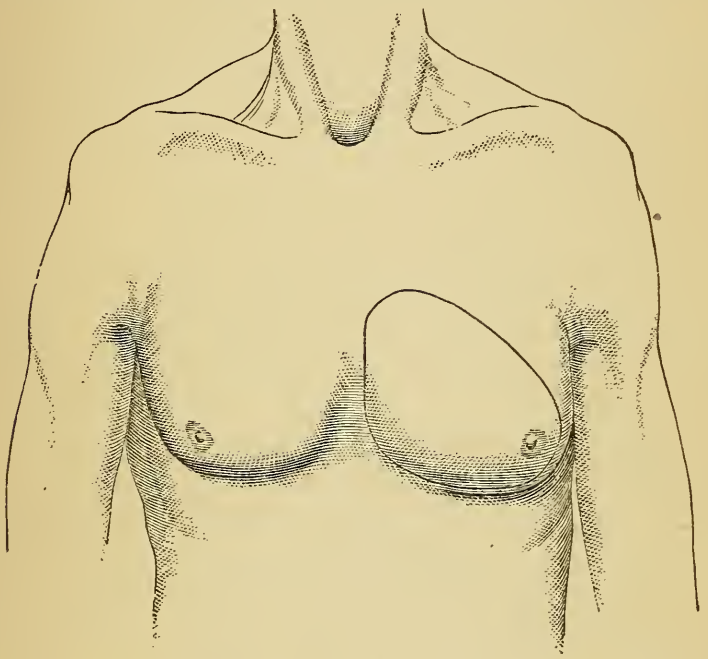
The results of mitral disease upon the system at large in-

clude congestion of the liver and kidneys as the most important lesions in the chain. The liver is enlarged, and may pulsate synchronously with the systole. Mesenteric congestion, with symptomatic indigestion from imperfect glandular functional activity, tympanites, ascites, hemorrhoids, are all incidents of this sketch. The renal congestion manifests itself by albuminuria, followed by anasarca, which commences in the feet and legs; a reverse of initial kidney dropsy, which begins in the cellular facial tissues. General anasarca is succeeded by effusions into the serous sacs, ascites, hydrothorax. Hydrothorax is either bilateral, or, if unilateral, it is usually disposed upon the left side of the chest, perhaps, because the enlarged heart presses upon the internal thoracic veins on the left side. Since both hepatic, mesenteric and renal congestion is evidenced by symptoms—query: what are the symptoms of pulmonary engorgement? The reply is, intercurrent attacks of hæmoptysis. Moreover, the pulmonary congestion is a fruitful harbinger of catarrhs, excited by atmospheric influences; so we add to the list bronchitis, more or less grave, as a frequent complication. Later in case œdema of the lungs occurs, as little by little the hepatic and renal activity is reduced, and the circulation is stored with imperfectly elaborated or eliminated nitrogenous materials; the sufferer is consequently liable to any form of serous inflammation, and finally to a possible death from uræmia.

Terminations of Mitral Regurgitation. The grave symptoms linked with mitral disease indicate that its duration must be largely measured by the number of complications, and the rapidity of their advent. Death, it is true, may suddenly terminate the scene, as the result of cardiac failure, but more frequently the patient succumbs to some of the incidental complications. The procession of complications is influenced by the avocations of the patient, the

presence or absence of syphilitic poisoning, the alcoholic habit, or the recurrence of endocarditis. The severity of the lesion, of course, has a primary and potential influence. Age is also a factor of peculiar import. Even serious mitral lesions in the very young are modified and sometimes vanish as maturity approaches.

Inspection and Palpation in Mitral Regurgitation. These



AREA OF CARDIAC DULLNESS IN A CASE OF MITRAL REGURGITATION.
(From Photograph.)

methods can be practiced simultaneously. The apex cardiac impulse is concentrated and lower than normal, but not usually so low as in disease of the aortic valves. When dilatation exists the impulse can be distinguished more outward and to the left than downward. The impulse is diffused, often undulating, because more of the ventricular wall impinges against the chest.

Percussion in Mitral Regurgitation. There is never as much hypertrophy as in aortic disease, but dilatation occurs early. The transverse dullness is increased in proportion to the dilatation of the left ventricle; the vertical in proportion to the hypertrophy.

Mitral Obstruction. *Auscultation.* The auriculo-ventricular valvular orifice may also be roughened or obstructed. The form of obstruction is so uniform that it has suggested the possibility that the lesion is congenital, rather than the result of endocarditis. The question is plausible enough to lead one carefully to study the history of each case of mitral obstruction. There is no doubt, however, that, in common with other lesions, it usually arises from endocarditis.

The shape of the mitral orifice merits special notice. The average circumference of the normal orifice is about four inches; the form is oval, with correspondingly narrow long diameter. In disease, this orifice is seriously modified. The chorda tendineæ and the valve may be thickened, stiffened into a rigid mass, and the leaflets fused so as to form a more or less conical tube, its smaller extremity opening into the ventricle; sometimes the orifice is extremely small, slit-like or "button-hole," admitting only the little finger, or even a pen handle through it. The valve may be encrusted with calcareous salts causing roughening of the auriculo-ventricular orifice without much obstruction. The effect on the auriculo-ventricular aperture is, of course, opposite to that of regurgitation. Presystolic murmur is associated with both stenosis and roughening, but is best developed if stenosis exists.

Dr. Flint has very concisely set forth the cardinal points connected with pre-systolic murmur, as follows:—

"(1st.) There are two varieties of murmur, rough and soft.

"(2d.) The rough murmur is vibratory or blubbering. It is imitated closely by producing with the expired breath sonorous vibrations of the lips or of the tongue. The softness of the second variety is bellows-like, resembling that of other soft cardiac murmurs. This murmur may vary in pitch and intensity, but as a rule it is low and weak.

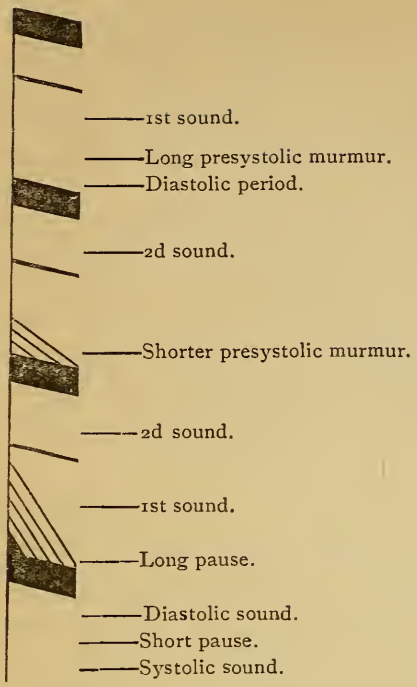
"(3d.) The rough murmur is due to the vibrations of the curtains of the mitral valve caused by the passage of the blood from the auricle into the ventricle. The soft murmur, like other bellows murmurs, may be due to contraction of the orifice through which the blood passes, or to the roughness of the surface over which it flows.

"(4th.) A rough presystolic murmur in general denotes a mitral obstructive lesion; the obstruction due to adhesion of the mitral curtains leaving a contracted orifice, the curtains remaining flexible. A presystolic soft murmur denotes either a contracted orifice, or roughness of the endocardial membrane.

"(5th.) A rough presystolic murmur exceptionally is produced when there is no mitral lesion, aortic regurgitation existing whenever the murmur is thus produced. The production of this murmur without mitral lesion may be explained by the physical conditions incident to aortic injury taken in connection with the mechanism of the murmur.

"(6th.) A rough presystolic murmur is not always present in connection with contraction of the mitral orifice, and by reference to the physical conditions, together with the mechanism of the murmur, its absence in certain cases may be satisfactorily explained."

The time or the rhythm with which the murmur occurs has been variously designated by the terms, presystolic, auriculo-systolic, post-diastolic, or simple mitral obstructive murmur.



The murmur may include the entire diastolic period, if the obstruction is extreme; on the other hand, it may only occur at the end of the diastole, immediately before the systole, thus meriting two of the terms, post-diastolic or presystolic. Custom has applied the term diastolic to the murmur of aortic regurgitation; in reality this murmur occurs at the commencement of the diastole, so the best term for the murmur of mitral obstruction or roughening is presystolic. The facts are, that at the end of the diastolic period both auricles contract, to express from their cavities the last remnants of blood and complete the filling of the ventricles. If mitral obstruction or roughening exist, it is evident that the murmur thereby generated will occur just before the systole, before the apex or carotid impulse, after the second sound. The murmur has in most cases two centres of equal intensity. The one corresponding with the auricle, the other with the apex, to which it is carried by the blood current. It must be remembered that at the time the murmur is produced the apex is not applied to the chest wall until the systole occurs. Perhaps this explains the fact that the murmur is best heard a little within the line of the apex beat; whereas in mitral regurgitation with systolic murmur, the sound is conducted to the ear when the ventricle is in approximation with the chest wall. A mitral murmur may some-

times be heard just outside the apex, because the ventricle may be both hypertrophied and dilated. Presystolic murmur is localized, and is not often transmitted from the apex, because the blood current is immediately diverted into the aorta by the ventricular systole. Thus it happens that the murmur is, as it were, clipped or cut off short at the apex.

In regard to the cases in which there is a murmur with a centre of maximum intensity over the auricle, we must remember that in health the left auricle is placed beneath the right, so that in cases of simple roughening of the auricular surface of the mitral valves the murmur may not be loudly heard over this cavity. But if true stenosis exist, one of the earliest consequences will be the enlargement, by hypertrophy and dilatation, of the left auricle, and the murmur will then be heard over the area included by the auricle.

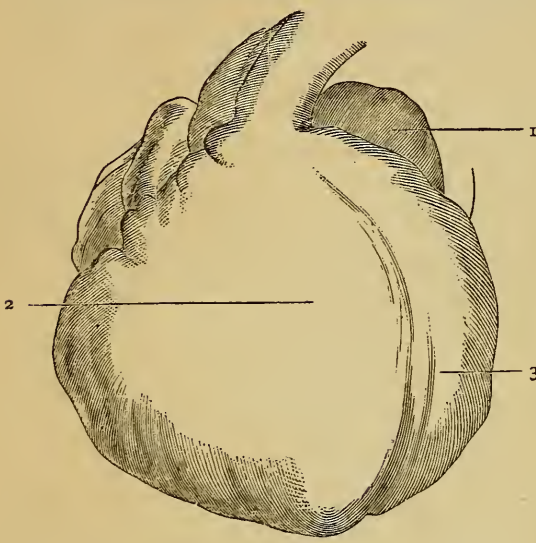
Concerning the exceptional transmission of presystolic murmurs into the axilla, or posteriorly, as reported by Dr. Andrews,* we recognize that such transmission is a possible, but an unlikely event. In his cases there may have been pulmonary consolidation, or pleural adhesions, or the murmurs may have been exceptionally loud.

In mitral obstruction there is always a marked accentuation of the pulmonary artery second sound, due to increased blood pressure in the pulmonary artery. The first sound may also become so valvular that it can easily be confounded with the pulmonary second sound, were we not guided by the ventricular impulse. Two explanations of this accentuation of the first sound may be offered. By one, we note that the blood pressure in the right ventricle is increased relatively with the elevation of the blood pressure in the pulmonary circulation, so that the tricuspid valves close with an intensified sound, audible over both ventricles. By the other hypothesis, if the obstruction be extreme, very little blood reaches the cavity of

* St. Bartholomew's Hospital Report, 1877.

the left ventricle at the time of the systole. We must, therefore, subtract from the first sound the blood element, and a portion of the muscular element, leaving either a valvular sound, as heard in typhoid fever, or a dull, blurred sound, as the valves may be much more thickened than roughened. While the first sound is often accentuated, it is also frequently irregular, without sufficient cardiac degeneration to account for the irregularity.

Reduplication of the first and second sounds is an occasional concomitant, explicable by the fact that the overfilled right ventricle and pulmonary artery lead to right ventricular, or pulmonary artery systole, prior to left ventricular or aortic systole.



CARDIAC ENLARGEMENT IN MITRAL OBSTRUCTION.

1. Enlarged Left Auricle. 2. Enlarged Right Ventricle. 3. Left Ventricle. Drawn from Specimen.

Disappearance of Presystolic Murmur. In certain instances the murmur of presystolic rhythm may vanish, to reappear, perhaps, in a few days. In these cases the auriculo-ventricular opening is apt to be very small, and the thickened valves are not traversed by sufficient blood to occasion enough vibration to produce a murmur. Position also materially influences the development of murmur in mitral obstruction. It can be heard in the upright, but not in the recumbent posture; sometimes the reverse is true. Rest in bed may render a presystolic murmur inaudible.

Double Mitral Murmur. Mitral obstruction and regurgitation, with double see-saw murmur, is not infrequent. In these cases the presystolic murmur often merges into the systolic murmur, which is apparently prolonged.

Effects of Mitral Obstruction. Dilatation and hypertrophy of the auricle may become extreme, owing to long continued and serious mitral obstruction. The normal præcordial area may be filled by the dilated auricle, and the left ventricle may be correspondingly depressed and displaced. In some cases the enlarged auricle presents a tumor causing bulging of the upper part of the præcordial area. The hypertrophy and dilatation exceed the similar condition in mitral regurgitation. In mitral obstruction we often find very marked hypertrophy and dilatation of the right ventricle, and, for a time, better compensation exists than in mitral regurgitation.

OUTLINE OF CARDIAC DULLNESS IN A CASE OF MITRAL OBSTRUCTION.

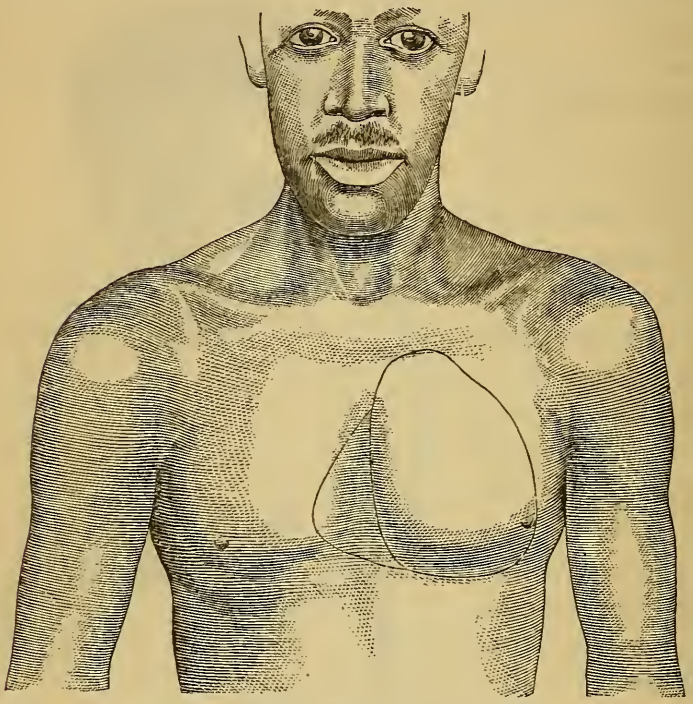
(From a Photograph of a child, æt. 8 years.)

Inspection in Mitral Obstruction. The outline of the left ventricle is normal. The præcordia is often enlarged over its upper portion and to the right of the sternum, instead of

the ventricle increased downward and to the left, observed in mitral regurgitation or aortic disease.

Palpation in Mitral Obstruction. Marked irregularity is a frequent symptom. Impulse at left apex feeble; impulse of right ventricle at xiphoid cartilage increased. When dilatation occurs, impulse becomes feeble.

Percussion in Mitral Obstruction. Transverse area of dull-



ENLARGED CARDIAC AREA IN A CASE OF MITRAL OBSTRUCTION.
(From Photograph.)

ness often increased to right of sternum and also above the fourth rib to the left of the sternum.

Terminations in Mitral Obstruction. Left auricular and right ventricular hypertrophy is more thoroughly compensatory than in cases of mitral insufficiency, and the compensation lasts longer. But transitory respiratory embarrassment, due to variations of the pulmonary circulation, is more fre-

quent than in mitral regurgitation. The associated dyspnœa becomes more severe if the compensative power is exceeded, because the lesion is, de facto, more grave. The pulmonary capillaries are continually overcharged with blood, so that slight increase in pulmonary blood pressure produces serious effects. Bronchitis of asthmatic type and cardiac asthma are frequent concomitants, and so is hæmoptysis and pulmonary congestion. The general series of phenomena outlined in the clinical history incident to mitral regurgitation find a parallel in the history of mitral obstruction.

Palpitation and Dyspnœa in Mitral Obstruction are more common than in other forms of organic heart disease. The causation is explained chiefly by venous repletion of the lungs and right ventricle, and partly by variations in the pulmonary vaso-motor tonus.

The Prognosis in Mitral Obstruction is controlled by the preponderance of hypertrophy over dilatation, and this is true of the prognosis of valvular disease in general.

The Pulse of both Mitral Obstruction and Regurgitation is apt to be small in volume, but in mitral obstruction the percussion wave is smaller than in mitral regurgitation, because there is less hypertrophy of the left ventricle. The pulse is also frequently very irregular in rhythm, especially during attacks of pulmonary congestion. The irregularity in these cases is probably due to a want of synchronous action between the right and left ventricles, due to the repletion of the former.

Tricuspid Regurgitation.* This is possible as the result of a primary lesion, but is more frequent secondarily, in consequence of (*a*) stenosis or obstruction of the pulmonary orifice; (*b*) valvular diseases of the left side of the heart; (*c*) from various diseases of the lungs, especially emphysema,

* Tricuspid stenosis is very rare. A valuable paper on this subject may be found in the *London Lancet* for April and May, 1881, by Dr. Bedford Fenwick.

hypertrophic or atrophic, and cirrhosis; (*d*) from simple dilatation of the right ventricle in consequence of fatty degeneration; (*e*) there may be regurgitation without valvular change, simply as the result of dilatation of the right ventricle, the leaflets of the tricuspid valve, which are normally competent to close the auriculo-ventricular aperture during systole, are withdrawn from one another by circumferential traction of the walls of the widened ventricle, so that their edges do not perfectly meet; (*f*) rupture of one or more chordæ tendineæ is possible. Tricuspid murmurs may be absent in cases where the valves are incompetent. Owing to the extreme dilatation of the walls of the ventricle, its contracting power may be temporarily or permanently exhausted, and the friction caused by the backward current of blood may be insufficient to cause a murmur.

Auscultation of Tricuspid Murmurs. The murmur is most loudly heard over an area two inches to the right of the xiphoid cartilage. It is transmitted upward to the base, and it may be transmitted toward the right axilla, especially if there is much dilatation of the right ventricle. It is not loud enough to be carried to the right axilla, because this murmur, as a rule, is very soft and blowing in quality. Tricuspid regurgitant murmur can be increased by exercise

Effects of Tricuspid Regurgitation. There is always dilated hypertrophy of the right ventricle and auricle. When there is much regurgitation there is more or less cyanosis, while, at the same time, the veins of the neck are much distended, especially the external and internal jugulars.

Venous Distention. "In healthy persons the internal jugular vein is not visible; the external jugular, on the contrary, is visible in the erect, if not in the recumbent, posture of the body. The external jugular vein usually possesses two sets of valves, one at its mouth and one in the middle of its course. The internal jugular is provided with valves at or a little

above its mouth. All these valves are very variable, both as to number and position. Moreover, they are often quite incompetent to close the vessel. This is especially the case with the valves in the internal jugular. The right internal jugular vein, right innominate vein, and vena cava superior, form a continuous channel, which is almost straight. For this reason all the signs about to be described are more marked on the right side of the neck than on the left, and they would be always more marked in the internal than the external jugular, were it not that the deep position of the former vein is a hindrance to observation."*

The distention of the descending vena cavæ and the jugular veins is not always permanent. If the cardiac action becomes more efficient the repletion of these veins may temporarily disappear. General anasarca, ascites, or pleural effusions may be expected. When there is tricuspid regurgitation there will usually be a venous pulse, direct or indirect. Indirect may be due to the fact that an over-distended ventricle bulges the tricuspid valves toward the auricle, and gives origin to impulse with each systole, which is measurably counteracted by the auricular systole. Direct pulsation occurs in consequence of a true regurgitation and propulsion of blood into the jugular veins through incompetent venous valves. Dr. Gee says that we can distinguish whether the valves are competent or not, by compressing the veins in the upper part of the neck, and observing whether they are filled with blood from below. The rise of blood and its pulsation from below is made characteristic by this method. The pulsations of the carotid artery may communicate systolic pulsation to the veins. The practice just mentioned, and a condition of the heart predisposing to distention of the right ventricle, are the best safeguards from error. The jugular veins are, of course, liable to overfilling from other causes, such as pressure by

* Gee. Physical Diagnosis.

pleural or pericardial adhesions, thrombosis, etc., acting on the vena cava or innominate vein. A permanent or long-continued repletion of the veins occasions their marked dilatation, most commonly of the right side, sometimes upon the left. Venous distention which has a pulmonary origin is possible. The descent of the diaphragm in normal inspiration is one of the motive powers of the circulation. In diseases in which this action of the diaphragm is interfered with, as emphysema, with chronic catarrh, the veins are always more or less distended, as a result of the pulmonary obstruction.

Inspection and Palpation in Tricuspid Regurgitation. The impulse of the right ventricle is extended to the right of the sternum. It is diffused, wavy and feeble. Systolic venous pulse in jugulars exists whenever there is much tricuspid regurgitation. Sometimes hepatic pulsations can be felt.

Percussion in Tricuspid Regurgitation. The cardiac area is extended beyond the right border of the sternum, and sometimes below it.

CHAPTER IV.

AORTIC VALVULAR DISEASE. OBSTRUCTION. REGURGITATION.

Aortic valvular lesions are associated with atheroma of the aorta resulting from inflammation, or with the fatty degenerative changes of advancing life. Aortic valvular lesions, however, sometimes follow rheumatic endocarditis. Roughening of the ventricular aspect of the aortic valves and stenosis are quite frequent as its sequel, and the changes in aortic roughening or stenosis are apt to induce valvular changes which cause regurgitation. The aortic valves are also especially subject to injury resulting from external violence. The lesions causing aortic obstruction or regurgitation can occur independently, or both may be met with in the same case. The murmur of aortic regurgitation is less common as an independent murmur, than that indicating aortic stenosis.

Aortic Stenosis or Roughening. *Auscultation.* The friction of the blood current as it passes out of the left ventricle develops a systolic murmur which is carried upward by the blood current to the place at which the aorta is most superficial, and is heard with maximum intensity at the second right costal cartilage. From thence the murmur is propagated into the carotids, and can in exceptional cases be heard even in the femoral, radial, or dorsalis pedis arteries. But aortic murmurs are also very widely transmitted, the sternum acting as a sounding-board, and aortic murmurs can be distinctly heard at the epigastrium, the apex, in the axillæ, or, when the murmur is rough or loud, at any part of the chest. When both mitral and aortic systolic murmur co-exist in the same case it is especially important to locate the point of maximum intensity of each murmur. Aortic systolic murmurs are synchronous with the apex beat or the carotid pulse. To dis-

tinguish them from a systolic mitral, we have the different location, and line of transmission with maximum intensity. Mitral systolic murmurs are not transmitted into the arteries of the neck; aortic systolic murmurs diminish in intensity as one auscults the apex after listening at the base of the heart or in the axilla. Two systolic murmurs are seldom of the same pitch and quality. Most frequently the aortic is the highest and roughest, since calcareous degeneration and atheroma are more frequent at this point.

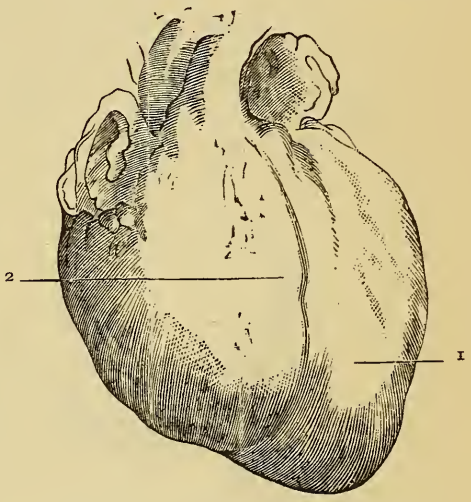
The pulse of aortic obstruction is sometimes of small volume (although the cardiac action is strong), if the obstruction is extreme. More often, however, the pulse is found to be full, strong and hard, an evidence of the ventricular hypertrophy.

Aortic Regurgitation. *Auscultation.* The diastolic murmur indicative of aortic regurgitation can be heard at the base, but is manifestly louder as the auscultator nears the aortic cartilage, which is the place at which that vessel is nearest the ear. Aortic regurgitant murmurs are transmitted in two directions. This happens because the blood current is flowing onward through the arterial circulation, and also by reason of the lesion, backward into the ventricle. In the latter case, the sternum conducts sound very readily, so that the murmur of aortic regurgitation presents a centre of equal intensity at a point just above the ensiform cartilage, near the mid-sternal line at the articulations of the sixth ribs. The murmur can sometimes be detected in the carotids, and along the aorta, but always with lessened intensity as the auscultator leaves the heart. Behind the sternum lies the bulk of the heart, and mostly the right ventricle, but at the apex the left ventricle is superficial, and aortic regurgitant murmurs can sometimes be heard at the apex.* Like aortic systolic mur-

* *See* record of two cases in which aortic regurgitant murmur was heard only at the apex. One was a case of rheumatic endocarditis; the other a case of valve rupture. Dr. Balthazar Foster suggests that a murmur heard

murs, diastolic aortic murmurs are distributed over a wide area, and in many cases can be heard in the axillæ of the right and left side.

The pulse of aortic regurgitation is very diagnostic. It has been called the trip-hammer pulse, or the Corrigan pulse. When the aorta is normally distended by the aortic systole, there is a gradual aortic recoil, or systole, which aids in forwarding the circulation. But if the valves are incompetent, the aortic systole forces the blood quickly backward into the ventricle, as well as forward through the aorta, and the arteries abruptly become abnormally empty. The ventricle is usually hypertrophied in aortic regurgitation; on this account the percussion stroke of the pulse resembles that of a ball striking the finger, and quickly rebounding. (*See* Sphygmograph.) I have found the Corrigan pulse in several cases of aortic dilatation without valvular lesion. The special character of the Corrigan pulse is much increased by elevating the arm above the head.

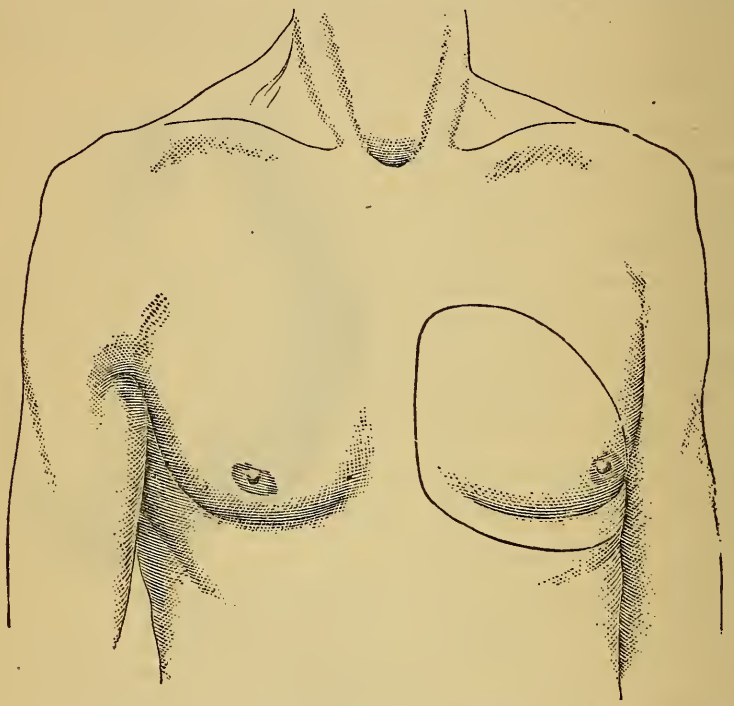


CARDIAC ENLARGEMENT IN AORTIC DISEASE.

1. Enlarged Left Ventricle. 2. Nearly Normal Right Ventricle. (Drawn from Specimen.)

as above indicates rupture of the posterior aortic segment. The other two segments being situated below the mouths of the coronary arteries; when they are diseased, the diastolic blood column is either prevented from entering them freely, or runs past their mouths into the ventricle again, and cardiac nutrition must be more directly affected, than if the other segment of the semilunar valve is diseased.—*Lancet*, Aug. 14th, 1869, p. 225.

Effects of Aortic Valvular Disease. The effects of aortic disease are observed chiefly in the condition of the left ventricle, in which there is always at first compensatory hypertrophy. The hypertrophy continues for a long period without being followed by dilatation. Old age is often attained by those suffering from aortic disease without much discomfort; but the advent of preponderating degenerative changes



AREA OF CARDIAC DULLNESS IN A CASE OF DOUBLE AORTIC DISEASE.
(From Photograph.)

is insidious and sure. To fully appreciate this, consider that in aortic obstruction the aorta is imperfectly filled, the coronary arteries which are supplied by the aortic systole (in its turn dependent on complete arterial distention), are imperfectly filled, and fatty degeneration slowly develops. The same changes take place in aortic regurgitation, but if the portions of the valves directly beneath the coronary arteries

are more perfect than other portions of the valves, the advance of degenerative changes is slower, since the blood is more or less efficiently directed into the coronary arteries: if the reverse prevails, or the mouths of the coronary arteries are more or less occluded, the course of dilatation and fatty degeneration is more rapid than in aortic obstruction. Another insidious source of danger is atheroma of the coronary arteries; this process begins as a consequence of increased aortic systole, which is caused by the violence with which the blood is forced by the hypertrophied ventricle into the aorta.

Terminations in Aortic Valvular Disease. In aortic obstruction the prognosis is excellent as long as hypertrophy is maintained; when dilatation occurs the symptoms of failing circulation, venous stasis, and dropsy soon follow. The same may be said of aortic regurgitation, but in this condition sudden death is more liable to occur than in any other form of valvular disease.

Inspection and Palpation in Aortic Valvular Disease. Impulse is displaced vertically more than transversely. If there is dilatation in excess of hypertrophy the displacement will be greater outwards than downwards. The impulse in the early stages is strong and concentrated. In the later stages it is feeble, wavy and diffused. Instead of the apex alone the ventricular wall impinges against the chest.

Percussion in Aortic Valvular Disease. In the early stages cardiac dullness is increased vertically; when dilatation occurs there is also transverse increase over the præcordia to the left of the sternum.

CHAPTER V.

AORTIC DISEASE; INCLUDING ANEURISM.

Lesions of the aorta are always the result of an increased arteral tension, usually of atheroma. The aorta is affected in a large number of cases of valvular lesion at the aortic orifice.

Atheroma of the Aorta. Of the physical signs of atheroma without much dilatation, there are none concerning which we can feel sure. Calcification of the arch is attended on auscultation with a rough systolic murmur which has a location of maximum intensity at a point between the sternal notch and the second right costal cartilage.* In differentiating systolic aortic valvular from systolic aortic murmurs without valvular lesion, we must remember that murmurs are heard with maximum intensity at that portion of the circulatory tract where the cavities in which the murmurs are generated approach closest the chest surface. Thus a systolic murmur becomes indicative of associated aortic valvular, with aortic lesion, when heard with equal intensity over the aorta as over the valves, *i. e.*, the second right costal cartilage; if the murmur is heard with maximum intensity over the aorta only, it is indicative of aortic lesion. In atheroma the aorta is unyielding, and the effect of its resiliency on the circulation is lost, and the heart becomes somewhat hypertrophied to meet the demand; but the heart is never so much enlarged as in valvular aortic disease. The general symptoms of atheroma of the aorta are pain from the pressure of the enlarged heart or aorta on adjacent tissues or nerves, palpitations of the heart,

* Murmurs indicative of atheroma, or aneurism, are sometimes telephoned into the smallest branches of the arterial system. They can be heard sometimes in the radials, or the dorsalis pedis arteries.

or angina, the latter doubtless symptomatic of changes in vaso-motor tonus. Atheroma of the aorta is accompanied by coincident atheroma throughout the vascular system; apoplexy and nutritive changes in the lungs occur as complications. Fatty degeneration of the heart is one of the important sequences of atheroma of the aorta or of the coronary arteries, and the symptoms incident to fatty heart are intertwined with the pathology of atheroma.

Aneurism of the Aorta. Aneurism may be classified as follows: (*a*) Simple dilatation of all the coats of the aorta; (*b*) Saccular Aneurism—forms of the sac various; (*c*) Dissecting Aneurism.

The first two conditions only will claim our notice in an outline of diagnosis of thoracic affections. Aneurismal changes are nearly always associated with, or preceded by atheroma, which by impairing the elasticity and contractility of the walls of the vessel interferes with its expansion and dilatation, even under the tension of the normal arterial blood pressure or this abnormally increased by any cause, such as occupation. The structural change is associated, in a major number of instances, with syphilis or alcoholism.

Using the word aneurism in its widest sense, as a local increase in the calibre of an artery, we observe that any form of aortic enlargement can give rise to some or all of the symptoms of aneurism, which are purely mechanical. The physical signs depend upon two conditions: First, the size and direction of the growth which determines the pressure symptoms and the results of inspection, palpation and percussion. Second, the nature of the sac, and the variety of orifice by which it communicates with the parent artery, are conditions which explain the results of auscultation.

Aortic Dilatation. *Percussion.* Practically, whenever the aorta is dilated there is dullness over the upper piece of the sternum, between the third rib and the notch; there is

also a broadening of the area of dullness laterally, encroaching materially upon the lung, usually to the right, but often also to the left of the sternal border. An excellent method by which to demonstrate this, consists in causing the patient to make full inspiration, noting that the percussion, previously dull, has become resonant; then cause full expiration to be accomplished, and note that the dullness is rendered more pronounced than it was in quiet respiration. The result is evidently due to the fact that the edges of the pulmonary lobe circle the aorta on full inspiration, and are withdrawn and the aorta exposed by expiration. The method of percussion is the same advised for examining the heart: commence in an interspace quite outside the area of impaired resonance; gradually approach the sternum until the area of impaired resonance is fixed.

Palpation in Aortic Dilatation. Thrill or fremitus may or may not be present; the sign is dependent on the roughness or smoothness of the internal wall of the artery. It should be sought for along the chest wall above the track of the vessel, and also in the sternal notch; and since the innominate and carotids may be also atheromatous, the thrill may be transmitted in them. Pulsation is another evidence, sometimes, but not always, present. The pulsation can be made more evident by causing the patient to incline forward, so as to bring the aorta as closely as possible in relation with the chest wall. Apply palpation, not only over the upper piece of the sternum, but also at the sternal notch. The aorta cannot ordinarily be felt over the sternal notch, but if it should be perceptible, there will be a jerking, inelastic impulse behind this point. Palpation will include the study of the pulse. There will be often associated atheroma of the radials more or less marked; the qualities of hardness, suddenness, jerkiness, are very striking. In some measure the Corrigan, or pulse of aortic reflex, is simulated; "even the hooked

apex of the sphygmographic tracing of aortic insufficiency is found in the rigid pulse of atheroma and calcification" (*Walsh*).

The orifice of the innominate or pulmonary artery may be more or less occluded by spiculæ of calcareous matter, or in extreme cases the lengthening of the tube due to the disease may reduce to a slit-like form the orifices of the arteries above named. On this account the pulse may vary materially in volume on one or the other side.

Inspection in Aortic Dilatation. By this method we may sometimes recognize local bulging of the tissues, forcible impulse of the carotids, and supra-sternal pulsation. Finally, some of the pressure symptoms to be alluded to under saccular aneurism may be present, but in a modified degree.

Saccular Aneurism. The tumor may grow in almost any direction ; its walls may be of various thicknesses ; sometimes they may become reduced to the thickness of the integument and the remains of the external coat of the vessel. The contents of an aneurismal sac may be a quantity of loose coagula or a laminated clot.

Inspection, Palpation and Percussion in Saccular Aneurism. These methods may enable us to outline a tumor. The success in determining the outline depends upon the direction of the enlargement. The tumor may occasion dullness which may be detected posteriorly as well as anteriorly. Pulsation in the course of the aorta is an important physical sign. A thrill can be detected with readiness proportionate to the thickness or thinness of the walls of the sac.

Auscultation in Saccular Aneurism. Murmurs may be systolic or diastolic, or both, in the same case. It is probable that the entrance of blood into an aneurism may be simultaneous with the cardiac systole, the exit of the blood synchronous with the second sound. Usually the murmurs have the hollow ring called bruit, analogous to the cavernous tone

imparted to respiration when the air enters a cavity in the lungs. The bruit is a hollow, reverberating murmur, usually longer than the other murmurs thus far described. Whether the murmur be single or double depends almost entirely upon the nature of the orifice. A murmur, when very prolonged, systolic and muffled, indicates that the tumor communicates with the aorta by a very small opening; or else that the tumor is largely filled with clots of fibrine, so that the orifice of a sac communicating with the aorta may be reduced to a mere depression. Aneurisms may exist without a murmur, but with only a dull impulsive impression, systolic in time. In some cases, if the tumor be superficial, the pressure of a stethoscope may influence the lumen of the aorta, and so develop a murmur.*

The second sound in aneurisms may sometimes be natural or accentuated. The heart may be normal or hypertrophied. The pressure symptoms are numerous and important.

Aneurisms of the Abdominal Aorta. The same methods of examination are applicable, and the same evidences are diagnostic as in thoracic aneurism. As additional points, notice that aneurisms of the abdominal aorta are often accompanied by an expansile pulsation, which can be observed in thoracic, but not so conveniently as in abdominal aneurism. Care must be observed not to confuse a transmitted pulsation imparted to a tumor in the abdominal cavity. The hand and knee position will obviate, in many cases, a transmitted pulsation, but an aortic enlargement will continue to pulsate distinctly. In abdominal aneurism the position upon the face or side gives more ease than the recumbent posture on the back. This is because the throbbing pressure of the tumor upon the vertebræ is mitigated, and this pressure is a source of great pain. The posture becomes a great aid

* As representative of many cases of aneurism without murmurs, *see Medical Times and Gazette*, Jan. 31st, 1880 (Broadbent).

to diagnosis in cases of pain in the back in which aneurism is suspected. In some cases lancinating pains run round the abdomen to the anterior parietes, giving the sensation of a cord around the body. A throbbing pulsation of the lower part of the abdominal aorta is a symptom not infrequently met with, especially in hysterical women.

Auxiliary Methods for the Study of Aneurism. We wish now to indicate some of the collateral evidences of aneurism and a method of study. In methods of investigation, the question of etiology is fundamental as a predisposing condition. Laborious avocations act the part of exciting causes second to the predisposing conditions already cited. The pressure symptoms occupy a leading place in view of the already determined etiology. The etiology presupposes arterial disease, and the location of the pressure symptoms will usually suffice to differentiate aneurismal pressure symptoms from those of other tumors. Saccular aneurism is a disease of early life; it chiefly occurs between the ages of twenty-six and forty years, after which the lesion will oftener be of the variety known as equable dilatation of the aorta. Pressure symptoms are determined by the location of the growth; in thoracic aneurism, if the tumor enlarges inward, the pressure symptoms upon the œsophagus or trachea, are noticed. The vertebræ may undergo atrophy, or necrosis; the intercostal, the pneumogastric, the sympathetic ganglia in the neck, may also suffer from pressure. In the latter case the irritation may occasion dilatation of the pupil of the affected side. The laryngeal nerves are involved, especially the right, which curves around the aorta, and laryngeal irritation or paralysis ensues. A strongly important point, even in cases where there is but partial paralysis, is the harsh laryngeal bark, which is very pathognomonic of laryngeal irritation, and is heard in tumors of the larynx, but should always lead to examination of the aorta. If the larynx is lifted upon the

forefinger inserted in the cricoid under the thyroid cartilage, we can often feel pulsation communicated to the finger by the enlarged vessel. There is one pressure symptom cardinal in its importance; this symptom is pain. It is persistent, and yet it can be mitigated by changes of attitude, which indicates that the pain has a mechanical origin. To illustrate this point, we have in the case of abdominal aneurism very marked mitigation of the pain when the prone posture on the face is assumed. In thoracic aneurism, if the sac impinges upon the trachea the patient steadily keeps the head forward, or perhaps sideways, or throws back the head while partly sitting in bed, in the effort to relieve pressure upon the windpipe. Pain is caused partly by necrosis or atrophy, the result of pressure upon the sternum or vertebral column; partly by pressure or irritation of adjacent nerve trunks, which gives rise to neuralgia. The proposition then is, that the diagnosis of aneurism requires the correlation of the etiology, the pressure symptomatology, and the physical examination.

Terminations in Aneurisms. Solution of continuity of the walls of the sac may be effected by slow perforation or sudden rupture. A sudden rent through the thinnest portion of the sac may be instantly fatal, by hemorrhage. Or, the stratified layers of the aneurism may be perforated by a very minute stream of blood, and several hemorrhages may occur, preceding the final issue by a variable space of time. There is hardly a conceivable situation at which hemorrhage has not occurred. If internal rupture happens, the pericardium, the pleuræ, the mediastinum, the stomach, the abdominal cavity, all may receive the contents. The blood may be voided by the mouth if hemorrhage occurs into the trachea, the bronchi, the lung substance, or the œsophagus. A most interesting fact in this connection is, that hæmoptysis from aneurism has been mistaken for the hæmoptysis of phthisis; of course, in

such cases the rupture of the sac occurs by slow perforation into a bronchus. Death in aneurism may occur from exhaustion from pain, insomnia, or the dyspnœa and asthma due to broncho-tracheal pressure.

Aneurism of the Pulmonary Artery. This is very rare, probably because the antecedent of aneurism (atheroma) is not likely to occur in this vessel. The usual signs of aneurism, pulsating tumor, thrill, murmur, must be expected, but there may be serious evidence of venous repletion. The differential diagnosis includes aneurism of the aorta, or an hypertrophied or dilated right or left auricle.

Pulsations of the Arteries without Aneurism. In the second left intercostal space, close to the sternum, one can sometimes find a pulsation which is diastolic in rhythm. The pulsation arises in the pulmonary artery, and is due to the repletion and distention of that vessel, and consequent exaggerated recoil. It is sometimes a feature of hypertrophy of the right ventricle, in its turn a sequel to mitral obstruction or regurgitation. A diastolic impulse in the right second intercostal space, close to the sternum, is a symptom of aortic dilatation.

CHAPTER VI.

INORGANIC OR FUNCTIONAL MURMURS IN THE HEART. THE VENOUS SYSTEM. THE ARTERIAL SYSTEM.

We have still a class of murmurs not grouped among those already described. They include functional or inorganic murmurs, venous murmurs, and arterial murmurs, developed with or without atheroma.

Inorganic murmurs are sometimes developed in the heart in the course of the zymotic fevers, in chronic Bright's diseases, in leucocythæmia—in a word, in any of those conditions in which the crasis of the blood is seriously reduced. There is ground for the belief that these murmurs are associated with the quality of the blood tissue, since they disappear as soon as the anæmia is overcome. They are also sometimes developed by coagulation of blood within the heart. The recognition of inorganic murmurs involves the following considerations: they are not associated with as much hypertrophy or dilatation of the various cavities of the heart as organic murmurs, and their centre of maximum intensity is variable.

Auscultation of Functional Murmurs. These murmurs can sometimes be heard at the apex, sometimes at the ensiform cartilage, sometimes above the third rib at the right or left border of the sternum. They are commonly heard most distinctly at the base, at which point the valves are situated in near relation with one another, and it is also the point nearest the origin of both pulmonary artery and aorta.

Diagnosis of Functional Murmurs. They occur in direct association with the etiological states to which they owe their existence. Inorganic murmurs, while they may be transmitted into the arteries, are often reinforced in intensity as one

recedes from the heart. If one causes the patient to turn the head to the side opposite the one examined, a humming-top sound, or venous murmur, the *bruit de diable*, may be heard. This makes it very probable that the arterial sound is hæmic.* Finally, the quality of the murmurs is soft and blowing. Sometimes they occur as a humming noise, and are always systolic. In regard to the possibility of thrombi developing a murmur, the same points will apply, save that their etiology does not require the association of the anæmic state. In thrombosis also, the sudden advent of murmur and the shifting location of maximum intensity are two valuable points. (*See* Thrombosis.)

Venous Murmurs. When the stethoscope is applied to the neck, a *continuous*, distinct humming sound is often to be heard. It is developed in the veins of the neck or the descending cavæ. This venous hum can be heard most plainly in persons who are profoundly anæmic; also in cases of inflammation of the venous coats, or pressure on a vein. The causes of pressure are chiefly pleuro-pericardial adhesions, or tumors of the mediastinum. The position of the body has much to do with the production of murmur, and it has been suggested that murmurs should be best heard in the erect posture, since the blood current is then at its swiftest. It may also be louder during inspiration, since this act markedly accelerates the flow of the blood toward the chest. These murmurs can be developed in a perfectly healthy person by pressure of a stethoscope.

Arterial Murmurs. These are developed solely by such pressure as can diminish the calibre of an artery, and so impede the blood current. The most important is the so-called

* Dr. Balfour believes that hæmic murmurs in chlorosis can be heard in the second left interspace, from one to two inches from the sternum. He believes that these are produced in the appendix of the left auricle.—*Edinburg Medical Journal, 1882.*

subclavian murmur, which can be heard in cases of incipient phthisis, in the subclavian arteries. It is usually a blowing, somewhat high-pitched murmur, yet one which is rather soft than harsh in quality. It is due to the pressure exercised by early consolidation upon the calibre of the artery, or it may be due to the pressure of pleural adhesions, which sometimes accompany early phthisis. Arterial murmurs due to atheroma can sometimes be heard in the superficial arteries, especially in the subclavian, axillary, or radials. It is possible that in states of reduced arterial tonus, arterial bruit can occur from the simple vibration of the walls of the vessel after the cardiac impulse, causing murmur.

CHAPTER VII.

FUNCTIONAL HEART DISEASE. VARIATIONS OF RHYTHM. NEUROSAL DISEASE.

In treating of the diagnosis of neurosal functional disorders of the heart's action, a diagnosis by exclusion of the diseases of the heart already enumerated, should be a primal step. The symptomatology is various, the etiology obscure. Insignificant causes broaden out into serious functional inefficiencies, as excessive indulgence in tobacco, in alcohol, in sexual appetite, and many psychical influences, profoundly influencing both vaso-motor tonus and the innervation of the heart. Many of the symptoms of "heart starvation," or fatty heart, may be intertwined with functional disorders without demonstrable cardiac lesion. In the group of functional disorders we shall allude merely to deviations of rhythm, and the symptoms of dyspnœa and pain.

Deviations of rhythm include intermission and irregularity.

Irregularity of rhythm and intermittency are conditions which differ only in degree. In intermittency the pause between the pulses is longer, there being an interval equal to that occupied by a pulsation. Irregularity is a want of accord in the action of the layers of muscular fibre of which the heart consists. The heart does not wait for a whole beat, as in intermission, but alters its rhythm irregularly. If the right heart does not complete its contraction at the same instant as the left heart, then the action may be reduplicated. Sometimes the periods of action and rest cannot be discriminated, and only a tumultuous throbbing is recognized. Patients sometimes describe this as a fluttering action of the heart. These conditions exist independently of organic disease. For instance, psychical causes, strong emotion by

terror, anxiety, grief, pain, fatigue, indigestion, use of tobacco, sexual excess, may cause irregularity, or it may be congenital. On the other hand, these variations of rhythm are very representative of fatty degeneration, or dilatation. The import of these variations of rhythm is chiefly based on the position and strength of the impulse as felt over the præcordia. If notable feebleness of impulse co-exists with irregularity, and insufficient systole, the import is serious, since it may indicate fatty degeneration and dilatation of the left ventricle, in which case the ordinary stimulation of the blood with which the auricular systole supplies the ventricle is inadequate, and a pause occurs until a second or third contraction supplies it with more. Irregularity is serious if associated with valvular disease. An irregularity deduced from causes which are neurosal is scarcely affected by effort—the pulse may quicken, but its irregularity is often rather diminished than quickened. When the irregularity is due to valvular disease, or fatty degeneration, a very slight effort, a brisk walk up and down the room, noticeably increases the irregularity.

Reduplications. The heart sounds may be doubled or repeated. It is a feature of intermittence, the sounds being reduplicated with some beats, and not with others. The normal sounds depend on the synchronous closure of the tricuspid and mitral, or the aortic and pulmonary valves. The synchronism depends very much on the blood-pressure in the cavities of the heart, or in the aorta or pulmonary artery. The first sound is reduplicated at the end of expiration or beginning of inspiration; the second, at the end of inspiration and the beginning of expiration.* In reduplication usually only the first or second sounds are reproduced. The condition

* For review of theories of causes of reduplication, *see* Dr. Sansom, on "Physical Diagnosis of the Heart;" also *Medical Times and Gazette*, 1877, on "Reduplication of Heart Sounds," by Dr. Barr.

must not be confounded with mere ineffectual systole, in which several entire revolutions of the heart occur, until sufficient arterial tension has been gained to produce a pulse; in this case the pulse at the radials and the heart beats do not correspond. Dr. Hayden groups the causes of reduplication as follows: "with simple neurosal functional derangement of the heart, or accompanying anæmia; with attenuation and weakness of the ventricles in persons of middle age, of nervous temperament; with a weak degenerating heart, and dilated atheromatous arteries; with simple hypertrophy of the ventricle."

"Guttman says it may occur temporarily in perfectly healthy persons; it may be noted at times in diseases of the heart, sometimes connected with mitral, sometimes with tricuspid disorder, but cannot be said to be characteristic of any particular affection." It may be a symptom in the course of typhoid fever, and it is a very common symptom in cases of mitral obstruction. Further, adherent pericardium leads to reduplication. The second sound is much more frequently reduplicated than the first; reduplication of the first sound is rare.

Palpitation is a disorder of rhythm, and an evidence of a spurt of the heart to accomplish its work. It is a symptom which may indicate both organic and functional disease. The essential pathology is probably a variation in the intra-cardiac blood-pressure induced by venous repletion of the lungs, or by variations in the arterial vaso-motor tonus. The vaso-motor tonus is profoundly influenced by causes operating upon the nervous system directly; for instance, the excitements of business, emotional, psychical disturbance of any kind. Functional digestive disorders have an important influence upon the cardiac innervation, and can induce palpitation. Nitrogenous food is taken up by the liver, and therein broken up from the form of peptones into glycogen, and the products of the urea series. The excess beyond the

purposes of nutrition cannot be excreted by the kidneys as rapidly as they are supplied by the liver. In the blood-vessel system these materials act as irritants, producing a recognizable increase in the blood-pressure.* Dr. Parks has performed some experiments relating to this subject upon the soldiers at Netley Barracks, England. In those experiments it was found that if nitrogenous food was withdrawn from their diet, the arterial pressure was sensibly reduced; on the other hand, it became elevated just in proportion to the increase of those articles of food containing nitrogenous substances. If in addition, renal activity is deficient, elimination of nitrogenous material is reduced, and the embarrassment of the circulation is increased. This is often the explanation of the temporary dyspnœa which so often occurs when the cardiac or pulmonary condition seems inadequate to explain the grave symptoms. Drs. Albutt and Johnson have assigned essentially the same explanation for the transient dyspnœa attending cases of nephritis, in which there is no dropsy to account for the condition. It has been called by them uræmic asthma, but the theory may be applied to many cases of functional palpitation.

Palpitation is a frequent incident in the history of simple hypertrophy, and attracts the patient's attention, because the enlarged heart gives origin to a forcible impulse. It is, however, a very frequent symptom of functional neurosal cardiac palpitation. It occurs in degenerations of the heart muscle, and among valvular diseases it is especially frequent in mitral obstruction.

Dyspnœa is a frequent symptom of cardiac disease, both functional and organic, but is also a prominent symptom of various pulmonary processes. An excellent test applicable in differential diagnosis, is the effect of exercise upon the symp-

* Lumleian Lectures, 1877: also *British Medical Journal*, September, 1877.

tom. The patient may breathe readily while quiet, but if requested to walk briskly or ascend the stairs, if cardiac disease exist, a more pronounced breathlessness comes on. Of course, an examination of the lungs should supplement the above evidence. In emphysema the dyspnœa is partly cardiac, partly pulmonary; an important point to be recognized from a therapeutic standpoint, since it indicates the use of both respiratory and cardiac stimulants for the relief of a patient. The position assumed by a patient for the relief of dyspnœa is significant. In cardiac disease the patient cannot usually recline, but must sit up during the paroxysms of dyspnœa.

Angina. Pain in the heart is a possible symptom of either acute or chronic heart disease. Pain may be complained of in any acute inflammation, such as endo- or pericarditis, or uneasiness may attend any form of chronic, functional or organic cardiac disturbance. Severe pain in the heart is sometimes felt by feeble or anæmic persons after unaccustomed exertion (*i. e.*, increased arterial tension). It may be a symptom of gastric indigestion, which may be referred to the heart, or pain may be brought on by excessive use of tobacco, or in the gouty state. Cardiac pain is differentiated from intercostal neuralgia by the painful spots in the course of the nerve in neuralgia, while pressure does not increase true cardiac pain.*

Angina Pectoris is a cardiac grip of phenomenal severity. The præcordial pain is most severe. The sensation has been described as though an invisible hand were grasping the heart, or it were being torn in pieces, and a feeling of impending death is experienced. The pain radiates down the left

* Pain with tenderness has been maintained elsewhere as a diagnostic symptom of intercostal neuralgia as separated from pleurisy.

arm, sometimes the right, shooting to the back or neck.* The heart palpitates somewhat, but its action is often slow and labored. The beat of the artery at the wrist may be small, irregular, or accelerated; or it may be full, strong and regular, not increased in frequency; depending on the condition of the cardiac and pulmonary substance. Again, there may be decided differences between the pulses, the left being almost or quite imperceptible.† The surface of the body is usually cold, and a clammy sweat beads the brow.

Summary of Diagnostic Symptoms. 1st. The attacks are paroxysmal, with long or short intervals, from a few minutes to an hour. 2d. Sense of coldness and cold sweat. 3d. Difficult breathing is not a prominent symptom. Cheyne-Stokes breathing may be present, but the breathing is always slow. 4th. The heart's action is not increased. 5th. The attack may pass off, or death may occur. The spells may be brought on by cold, fatigue, mental excitement, and are most dangerous; since the heart may pause in any paroxysm. Naturally there must be many gradations of intensity of attack, from moderate distress to anguish. The true anginæ are rare; intercostal neuralgias, rheumatism, reflex pain from dyspepsia, are common. The teaching of to-day seems to accord with the following statement: Angina pectoris is a

* "The pain of angina is distinctly located in or about the mid-sternum, whence it radiates. Eulenberg says this is due to the connection between the superior cardiac nerve and the anterior branches of the four upper cervical nerves, while the middle and inferior cardiac nerves are connected with the four lower cervical nerves, uniting in the brachial plexus and first dorsal nerve. The pain usually runs out at the peripheral ending of the ulnar nerve, especially the little finger. It is almost invariably found on the left side only. In a case where it was found on the right side the pulmonary artery was the seat of disease."—*Diseases of the Heart, Dr. Fothergill, London*, 1879, *p.* 285.

† Hamilton Osgood, *American Journal of Medical Sciences*, Oct., 1875.

disease associated with vaso-motor spasm, as proved by Dr. Lauder Brunton. "It is divisible into two classes; the *first* a class in which the arterial tension is only increased during the attack; the *second*, in which there is persistently high arterial tension, though still higher during the paroxysms—this class including the majority of cases." We are cognizant that arterial spasm is brought about temporarily by the introduction into the blood of imperfectly reduced albuminoids, or their imperfect elimination; conditions brought about by constipation, imperfect intestinal digestion, hepatic inefficiency. Also by deficient elimination consequent on changes in the cutaneous circulation, or the activity of the respiratory or renal functions. Angina is, however, seldom an uncomplicated nervous disease; mostly some more or less serious organic lesion prevails, and the weakened heart cannot adequately maintain the circulation. In such cases atheroma is an almost invariable concomitant; this lesion vindicates the second division of cases of angina. An imprudence in eating, or an exposure to cold, involving a checked cutaneous elimination, may easily increase arterial tension, and precipitate an attack.*

Since cardiac disease may develop atheroma, or atheroma may induce cardiac disease, we may easily have angina, a concomitant of any kind of heart disease. This hypothesis is abundantly confirmed by the post-mortem table. In conclusion, a study by the sphygmograph might indicate this great predisposing cause, increased arterial tension and its outcome, atheroma.

* In either class psychical causes may result in increased vascular tension. "John Hunter is reported to have said, my life is in the hands of any rascal who chooses to annoy or abuse me. He afterwards died of an attack brought on by his ungovernable temper."—*Da Costa's Diagnosis.*

CHAPTER VIII.

CARDIAC THROMBOSIS.

Intra-cardiac thrombosis occurs sometimes as the direct result of endocardial inflammation, but more frequently by feeble circulation of the blood from failing cardiac propulsive power. Slowing of the blood current through the lungs, but especially through the right heart, occurs in some organic lesions of the heart and pericardium and certain pulmonary lesions, especially pneumonia. Certain conditions of the blood also render it liable to coagulate. In post-mortems clots are frequently found in the heart; and in works devoted to pathology the difference between ante- and post-mortem clots are enumerated. The clot is usually on the right side of the heart.

Murmurs may occur in acute pulmonary disease, especially pneumonia, which disappear on convalescence. It is possible that they may be caused by thrombosis, or they may be tricuspid murmurs due to dilatation of the right ventricle, which may become replete with blood. In acute febrile diseases murmurs sometimes develop, and vanish when convalescence occurs. The exact pathology of these murmurs has not yet been settled.

Diagnosis of Thrombosis. The urgency of the general symptoms varies in proportion to the size of the clot. Dyspnœa is often marked. The cardiac rhythm may become very rapid and irregular, with rapid and irregular pulse, pallor, coldness, more or less tendency to syncope, and, if the clot be very large, all the consequences of venous congestion. The heart sounds may be dulled: if a murmur preexists it may be modified or suppressed, depending upon the location or distribution of the clot. Murmurs due to thrombi

develop suddenly, are usually systolic, basic, soft and blowing, not transmitted in the route of the blood current. The position and distribution of the clot varies, and consequently the location of maximum intensity of murmur frequently changes. Murmurs due to thrombosis are heard most distinctly over the superficial area of the cavity in which they are produced. The right ventricle is a favorite location for clot; therefore the murmurs will be distinct at the ensiform cartilage. In murmurs due to thrombosis the hypertrophies and dilations incident to special valvular lesions will not be observed, nor will the murmurs be transmitted in the direction of valvular murmurs, and their position of maximum intensity will be variable.

The results of thrombosis vary with the size and location of the clot. Small thrombi may undergo resolution and disintegration. We have seen cases where the diagnosis seemed to be established with a fair amount of certainty, in which recovery took place with a disappearance of the physical signs. A diagnosis of this sort is naturally subject to criticism or objection. Large clots formed suddenly destroy life in a few days, but death is partly due to the preëxisting disease. Where the formation of a clot is slow, life may be prolonged for an indefinite period.

Thrombosis of the Pulmonary Artery. The condition includes thrombosis or plugging of the main trunk or its radicles. It is due to right heart failure or is the result of embolism. If the main trunk of the vessel is involved, we may have sudden extreme dyspnœa, and death. If the clot be smaller or distributed further from the heart, the symptoms of dyspnœa vary in accordance with the efficiency of the collateral circulation. It may be possible, by excluding other murmurs, on grounds already explained, or by an analysis of the predisposing causes, to establish a blowing systolic murmur, distributed in the course of the pulmonary artery, indicative

of pulmonary artery thrombosis. Or, again, if infarction of the lung has occurred, we may expect to find the physical signs appropriate to condensed pulmonary tissue, signs which are appraised by their association with the predisposing causes of embolism.

Arterial Thrombosis depends on local disease of the arterial coats, upon pressure or obstruction from causes external to the vessel, and upon alteration of the blood crasis in which its coagulability is increased. These causes may be associated with diminished vaso-motor tonus, which favors a more sluggish circulation than natural. Embolism in any locality may be the outcome of thrombosis, and can be distributed in any part of the arterial system. In the brain the diseases of the arterial system favoring thrombosis produce increased brittleness, and also favor rupture and apoplexy. In venous trunks, in the radicles of the intestinal veins, ulcerations may originate by the agency of local inflammations (phlebitis), which may terminate in thrombosis, to be followed by embolism. Emboli from the intestines are often arrested in the liver, a fruitful cause of hepatic abscess, from whence fresh emboli may be carried to the heart. From the venous trunks of the extremities emboli may be carried to the right heart and lungs directly. It is possible for the emboli to pass through the pulmonary circulation, and obstruct some artery, but arterial embolism usually has its genesis in the arterial system.

CHAPTER IX.

DISEASES OF THE CARDIAC SUBSTANCE. HYPERTROPHY. DILATATION.

We shall study in this chapter the diagnosis of the lesions of the substance of the heart when these lesions are not directly connected with valvular disease.

Simple Hypertrophy, *or an increase in the thickness of the cardiac walls.*

It must be remembered that normally the walls of the right ventricle are thinner than the left, the proportion between them being as 1 to 3 or 4. The causes of the hypertrophic process originate in consequence of excessive ingestion of food or stimulants, or any excessive form of muscular exercise. Functional palpitation, if long continued, may induce hypertrophy. It is common in the gouty state, and in the forms of Bright's diseases in which interstitial inflammation predominates.*

Eccentric or Dilated Hypertrophy, *by increase in the cardiac walls, and dilatation of the cavities.*

In hypertrophy the form of the heart is retained, but this is lost in proportion as dilatation is superadded. Hypertrophy is nearly always combined with dilatation, representing a condition in which the capacity of the cardiac cavities is increased disproportionately to the thickness of the walls, whether the walls be thicker than normal, of natural thickness, or thinner.†

* The gouty diathesis is often associated with increased arterial tension, by reason of defective elimination, etc. Sometimes, even without recognizable renal changes, there is a marked tendency to hypertrophy.

† In valvular, as well as simple cardiac disease, we are accustomed to speak of the heart as hypertrophied (though dilatation is combined

This form of hypertrophy is a process often interlaced with fatty degeneration of the heart, and the physical signs of both conditions are often intertwined. Dilated hypertrophy may occur as a sequence of valvular disease, but it may also be an independent process. It may affect any of the cavities of the heart; but the ventricles in particular may be affected, especially the walls of the left ventricle. The right side enlargements are secondary to the left-sided diseases, or certain pulmonary lesions which provoke repletion of the right heart. The valves themselves undergo hypertrophy; the same condition may be observed in the chorda tendineæ. These lesions are very frequently observed in the tricupsid valve; sometimes in the mitral or aortic valves.

Inspection and Palpation in Simple Hypertrophy. In the young and those in whom hypertrophy has been of long standing, there is fullness and prominence of the præcordial space, indicating the size to which the heart has attained. The impulse of the heart is very forcible and distinct. The apex beat of the hypertrophied left ventricle is displaced downward and somewhat outward. The apex beat is a concentrated, localized, heaving impulse. The shape of the heart is retained, and the action of the heart is regular. The carotids and aorta often pulsate forcibly, and the pulse is indicative of arterial fullness and a strong vis a tergo. Normally the right ventricle is uppermost; but as the left ventricle is hypertrophied or dilated, it becomes superficial.

Percussion in Simple Hypertrophy. Cardiac dullness is increased downward and outward upon the left side of the sternum in left ventricular hypertrophy.

with it) so long as the hypertrophy enables the heart to maintain the circulation effectively. We say the heart is dilated when it loses the power to maintain the circulation adequately, although the heart muscle is hypertrophied. *See* Chap. I, Part II.

Auscultation in Simple Hypertrophy. The first sound in hypertrophy is prolonged and dull; the second sound is accentuated. Murmurs may be present when there is associated valvular disease.

The combination of these physical signs is essential to the diagnosis of hypertrophy, since excited action of the heart, in certain neurosal states, may simulate enlargement.

Hypertrophy and Dilatation of the Right Ventricle. These conditions usually occur together, consequent upon pulmonary emphysema, or left-sided heart lesions. The area of dullness extends beyond the right border of the sternum, over the cartilages of the third, fourth, and fifth ribs, and if the right auricle is dilated, the dullness extends from the second costo-sternal junction to the third; mostly behind the sternum. Concurrent signs are the pulsation of the right ventricle in the epigastrium, the venous turgescence more pronounced than in left-sided lesion, venous pulse, and perhaps pulsation of the liver. Systolic pulsation of the liver can be separated from cardiac pulsation in that region, principally by the wide area of pulsation. The phenomenon is rare.*

Dilatation of the Heart may correspond with eccentric hypertrophy and may be associated with valvular disease. In simple dilatation the walls of the heart may be normal in thickness, but the cavities enlarged; or the walls of the heart may be very thin, the condition being known as passive dilatation. The causes of this condition may be valvular disease, fatty degeneration, emphysema, chronic bronchitis, chronic rheumatism, or Bright's diseases. It sometimes occurs in early life in those of feeble resisting powers. The right side of the heart is more frequently involved than the left, in simple or passive dilatation. Dilated or eccentric hypertrophy is more frequent on the left side.

Inspection and Palpation in Dilatation of the Heart. A

* *See* Epigastric Pulsations.

cardinal point is, that in dilatation the pyriform shape of the heart is lost; instead, the form of the ventricle becomes sac-like. The impulse of the heart is carried downward as in hypertrophy, but more outward than downward. The pulsation striking the chest is not concentrated and localized, since it no longer represents the apex beat, but more or less of the body of the ventricle impinges against the chest; the apex beat is spoken of as diffused. The impulse is weakened in proportion to the degree of dilatation, and is felt as a short, feeble, and fluttering slap. The impulse may also be irregular; the outline of the ventricle can be seen to contract vermicularly. The volume of the pulse is small.

Percussion in Dilatation of the Heart. The sac-like or globular form, instead of the pyriform shape, is very noticeable in proportion as dilatation proceeds, the apex becoming more and more obtuse. The transverse increase in dullness can be found both over the area of the right and left ventricle.

Auscultation in Dilatation of the Heart. The sounds of the heart become clearer and sharper if the dilatation is moderate, the thinning of the walls of the heart seeming to aid the transmission of the sounds just as hypertrophy seems to muffle them. When the process of dilatation is advanced, and the walls become very thin, the sounds are apt to become proportionately faint and ill-defined. Irregularity may be noted with reduplication of the sounds. All irregular hearts are by no means dilated or fatty, but all cases of advanced dilatation or degeneration, are apt to be irregular. The cause of the irregularity is the reduced ability of the heart to cope with the difficulties of the circulation. The cardiac systole is unable to fill the aorta, imperfect aortic systole follows, and the coronary arteries are therefore imperfectly supplied. Moreover, the cardiac systole is incapable of emptying the cardiac veins, and fatty degeneration is favored.

General Symptoms in Dilatation of the Heart. There is a tendency to venous congestion of the pulmonary, the portal, the chylopoetic and renal circulation. Œdema, hæmoptysis, indigestion, constipation, jaundice and albuminuria, with scanty secretion of urine ensue. Anasarca manifests itself in the dependent portions of the body, or there may be pleural or abdominal effusions. Venous congestion predisposes to intercurrent catarrhs and coughs, which are aggravated if there is associated emphysema. Dyspnœa is always a more or less troublesome symptom. "In emphysema, atrophic or hypertrophic, the amount of dyspnœa on exertion will be the best guide to the structural state of the heart."

Enlargement of the Left Auricle. Normally the left auricle lies beneath the right auricle. If enlarged, it occupies a space transversely to the left of the sternum, outside the normal area of cardiac dullness between the second rib and the upper margin of the fourth rib vertically. The left ventricle may be very much depressed by the enlarged auricle, and may simulate a true hypertrophy. The lesion occurs especially in connection with mitral stenosis; with less frequency in mitral regurgitation. Percussion will define the tumor; auscultation will reveal the time of the murmur, which indicates the pathology of the lesion. Palpation reveals pre-systolic pulsation of the auricle in mitral obstruction, and systolic impulse in cases of mitral regurgitation. A convenient way of demonstrating the different centres of pulsation has been suggested. Cut two small circles of sticking plaster, about the size of a cent. Transfix the centre by a pin, so that the head is in contact with the adhesive side of the plaster; attach the plasters to the different centres of pulsation. The shafts and points project forward, and you have two levers which vibrate with the movements communicated to them by the several pulsations. These levers can be rendered more distinct by attach-

ing small pieces of tissue paper as standards. Diastolic pulsation of the pulmonary artery is possible as an evidence of repletion but the position for this pulsation is close to the sternum.

CHAPTER X.

FATTY DEGENERATION OF THE HEART.

This condition is often connected with the dilatations due to valvular disease, or with simple or passive dilatation, but may exist independently of either. When the blood tissue is imperfectly elaborated, or when it is inadequately depurated, the fundamental conditions of molecular life, *i. e.*, growth, development and reproduction, are impossible. We have already shown that the condition of dilatation of the heart favors impairment of circulation, and subsequent degeneration; all dilated hearts are not fatty hearts; but all fatty hearts, from weakness of their walls, soon become dilated. When fatty heart is developed independently of valvular disease, it is often due to atheroma. Fatty degeneration of the heart may be preceded by atheroma of the coronary arteries, but this is the expression of a more general atheromatous change, especially in the aorta. The imperfect crasis of the blood finds its expression in gout, in gouty bronchitis, in eczema, in muscular rheumatism, indigestion, neuralgia; all traceable to excessive nitrogenous waste in the blood. The excessive use of alcohol, prolonged wasting disease, notably prolonged suppurations, anæmia, are all predisposing causes. In fevers, especially typhoid fever, we have a rapid fatty degeneration, and wasting of the cardiac muscle, a peculiar incident of the disease; besides which Dr. Fothergill has described an acute fatty degeneration of the heart, which will be subsequently given in his own language. The essential etiological fact is that fatty heart is but a local incident of a widespread process.

Fatty heart, as a symptom of general tissue degeneration, is sometimes a disease of middle life, but mostly it occurs in

more advanced years, intertwined with arterial diseases, rigid, incompressible arteries, tortuous temporals, as the expression of fatty and calcareous degenerative change, almost normal to old age. We have no positive physical signs of the change, to be developed by a study of the heart. We may suspect it, says Dr. Da Costa, "if the signs of weak action of the heart, feeble impulse, ill-defined sounds, co-exist with oppression, with a tendency to coldness of the extremities, with a pulse permanently slow or permanently frequent, or irregular, and are met with in a person who is the subject of gout, or of wasting disease, or is very intemperate, or has arrived at a time of life at which all the organs are prone to undergo decay. Something more than a suspicion is warranted, if in addition there be proof of atheromatous change in the vessels, or fatty degeneration elsewhere, such as arcus senilis; or if it be ascertained that the patient suffer from pain across the upper part of the sternum, and from paroxysms of severe pain in the heart; that he sighs frequently; that he is easily put out of breath; that his skin has a yellow, greasy look; that he is subject to syncope or to seizures, during which his respiration seems to come to a stand still; and that he is liable to vertigo, or to be stricken down with repeated attacks having the character of apoplexy, save that they are not followed by paralysis." In reference to the pulse it may be fast or slow. Since the heart is weakened, the pulse rate may easily be slowed, just as when aconite or antimony depress the circulation. But if cardiac degeneration is extreme, the weakened muscle usually responds by a series of more or less abortive systoles, several failing to accomplish the results of one normal systole. The rate of the pulse is necessarily only a corroborative sign, since a very fatty heart may contract indolently, or accomplish the circulation by a series of abortive systoles. Exertion may precipitate an attack of palpitation, while the pulse may be very slow when at rest. So far as arcus senilis is an evidence of fatty heart, we must

recollect that there are two kinds of arcus. The arcus dependent on calcareous degeneration of age is recognized by a circle, or a semicircle, of opaque whiteness. If this be well defined, and the rest of the cornea bright and translucent, it is probably no indication of serious internal degenerative change. But the arcus associated with a blurred, ill-defined ring, if the ring be yellowish rather than white, the rest of the cornea slightly cloudy, the chances of cardiac degeneration are serious; at the same time fatty heart may be present without pronounced arcus of any kind. Fatty heart is not commonly associated with increase of the area of præcordial dullness, or serious dropsy, nor local congestions, unless associated with dilatation, but the conditions are often interlaced. Cheyne-Stokes breathing may occur in fatty heart. Dyspnœa on exertion is an important symptom, especially of right-sided fatty heart. In old persons, however, a moderate degree of dropsy, mostly of the lower extremities, is frequently a symptom of cardiac feebleness, and the urine may show traces of albumen. One always must exclude dropsy due to changes in blood crasis, impairment of vaso-motor tonus, which are not directly dependent on the condition of the liver or kidneys.*

Occasionally, in functional heart disease, but often in organic, deficient oxygenation of the blood has the effect of producing a state of drowsiness, or even mild delirium may supervene if elimination is deficient. If dyspnœa is an evidence of seriously deficient cardiac action, stupor may be added to the symptoms of these cases, which are usually associated with dropsy and albuminuria. The question of differential diagnosis between cardiac dropsy with uræmic symptoms, and renal dropsy with uræmic symptoms, is

* *American Journal of Medical Sciences* for 1871, vol. 2. *See* paper of Dr. H. C. Wood on "Acute Dropsy, Scarlatinal and Idiopathic;" also Trans. Phila. County Medical Society for 1879, '80, page 79. Anasarca as a symptom of deficient vaso-motor tonus, by Dr. E. T. Bruen.

often raised. The cyanosis, especially of the face, and the history that the dropsy began in the more dependent portions of the body, characterize the former case, while in the latter the face is white, the skin pallid, and the dropsy began in the face.

Dr. Fothergill has recently described a condition of heart starvation which might represent to us acute or subacute fatty degeneration versus the chronic condition. His language is as follows: "It is a condition springing from defective assimilation: all the tissues are badly nourished, but the heart and diaphragm suffer most from the ceaselessness of their round of duty. Physical signs of feeble impulse, and weak, ill-defined sounds, are identical with fatty heart. Heart starvation, independent of wide-spread changes, is associated with anorexia, indigestion, deficient hepatic secretion, pale stools, deposits in the urine, and the characteristic tongue. There is depression, irritability from the blood-poisoning of mal-assimilation, and the train of symptoms of deficient blood depuration we have already sketched. We may even have a species of angina. The disease may pass into chronic fatty heart through development of arterial changes." This cardiac state, he believes, occurs in early and middle life, associated with overwork and deficient digestion. Dr. Fothergill thinks that prolonged debauch, during which alcohol alone is taken, may result in actual fatty decay of fibrillæ, which wear out and are not repaired, because of the absence of albuminoids in the food. We are familiar with a similar fatty state in fevers with high temperature, a lesion which is repaired when the assimilative processes are restored. This phase of fatty degeneration of the heart is so frequent in typhoid fever, that it constitutes the cause of death in many cases of the disease, in which improper exertion is allowed. During convalescence, death from cardiac syncope, or organic mischief, may follow premature exertion. Acute fatty degeneration of the heart may

be followed by repair of the tissues if the causes which produced it disappear. The symptoms of the above condition may, however, be purely neurosal.

Cardiac Atrophy. In phthisis, in carcinoma, in constitutional syphilis, in suppuration of bone, and also in chronic adhesions of the pericardium, cardiac atrophy has been observed. Conjecture has referred this lesion to atheromatous disease, or obstruction of the coronary arteries. It is interesting to note that atrophy, with contraction and consequent diminution of the size of the cardiac cavities, is perhaps the condition which has been called concentric hypertrophy. In adherent pericarditis we have seen a specimen in which the walls of the left ventricle equalled an inch in thickness, but the cavity of the ventricle would scarcely contain a large walnut. There are no symptoms or signs by which this condition can be positively recognized. Palpitation, or other functional disturbance, even a frequent pulse, together with the recognized existence of the predisposing conditions, are the best guides.

TABLE OF DISPLACEMENTS OF HEART.

Upward.	By enlarged liver. By enlarged abdomen. By tumor, ascites or tympany. By pericardial effusion or adhesions.
Downward.	By enlargement of the right or left ventricle; or of left auricle. By tumor of the lung.
Laterally to left.	By dilatation of the heart—right-sided pleural effusion or pneumothorax; left-sided pleural adhesions. By cirrhosis of left lung. By pericardial adhesions.
Laterally to right.	Left-sided pleural effusions or pneumothorax; pleural adhesions may form and retain it on right side. By cirrhosis of right lung. By emphysema; right ventricle most displaced.

The influence of aneurism is various. On the whole, the heart is not usually displaced unless there is valvular disease. Diagnosis of congenital displacement is based on exclusion and history of the case.

CHAPTER XI.

THE PULSE. ARTERIAL TENSION. THE SPHYGMOGRAPH.

The value of the pulse as an aid in physical diagnosis has been already partially considered in the foregoing chapters. The variations in frequency in valvular disease and cardiac dilatation or degeneration are chiefly in the direction of increase. A slow pulse rate has been shown to accompany certain cases of cardiac degeneration, but an extremely slow pulse is even more characteristic of neurosal than degenerative cardiac disease. In hypertrophy the pulse is firm, full and bounding. The varying pulse rate in pericardial disease is a notable symptom of that lesion.

Increased arterial tension can be detected by auscultation and palpation, the latter method being supplemented by the use of the sphygmograph.

Auscultation discloses an increased arterial tension by the increased sharpness of the second sound (accentuation). It is heard at the second right costal cartilage, if the increased tension occurs in the arterial system; and at the second left costal cartilage when increased tension occurs in the pulmonary artery. The arterial tension is increased by several conditions: (*a*) by an increased cardiac action, or by hypertrophy of the heart; (*b*) by renal disease, especially the chronic forms, with interstitial as well as tubular changes; (*c*) in atheroma of the arterial system; or increased vasomotor tonus from neurosal causes.

It *is the persistence* of the elevated tension which is significant of structural change.* Repletion of the pulmonary

* Dr. Edes has reported cases, presumably in good health, in which a high grade of arterial tension, as shown by sphygmographic tracings, is demonstrated, but it is not stated whether the high tension was a *persistent* feature.—*Boston Medical and Surgical Journal of May 19, 1881.*

artery, with increased tension, occurs in both forms of mitral disease, often in emphysema, or in pleurisy with effusion. The arterial tension is decreased by (*a*) degenerations of the heart, or imperfect systoles; (*b*) by diminished vaso-motor tonus. In consequence, the aortic, or pulmonary artery second sound, is diminished in intensity.

FEEBLE HEART SOUNDS.	
First sound, enfeebled.	Advanced fatty degeneration; dilatation. Neurosal influences. Changes in diminished vaso-motor tension.
Second sound in pulmonary artery or aorta, enfeebled.	The above conditions. Also reduced vaso-motor tonus in aorta or pulmonary artery, whether from feeble cardiac systole, owing to degenerative, or neurosal causes.
Pericardial effusion is a prominent cause of the enfeeblement of the heart sounds.	

ACCENTUATED HEART SOUNDS.	
First sound, accentuated.	Mitral obstruction, possibly aortic obstruction and regurgitation Degenerations or dilatations of the ventricles.
Aortic second sound, accentuated.	Chronic forms of Bright's Diseases. Palpitation when the heart muscle is normal. Hypertrophy of the heart. Atheroma; increased vaso-motor tonus from neurosal causes.
Pulmonary second sound, accentuated.	Circulatory obstructions in the lungs. Pleurisy with effusion. Emphysema. Mitral valvular disease, especially mitral obstruction.

Palpation in the Diagnosis of Arterial Tension. We may note if the radial artery is normal in structure. In atheroma it may be rigid, unyielding, and a strong pulsation may be apparent, on account of the rigidity of the artery. If there is associated cardiac degeneration or dilatation the pulse may be feeble and the arterial tension low. In increased arterial tension the artery is less compressible than normal. The arterial tension is increased in inflammations, especially in inflammation of the peritoneum, but in these cases the volume of the pulse is small and is termed wiry. In acute diseases of robust persons the pulse is hard and full. Caution must be observed not to confuse it with the gaseous or full pulse of an artery which has lost its tonus. The gaseous pulse, though full, is compressible, and the percussion impulse is weak, proving that a full pulse and a strong pulse are not synonymous. The pulse of diminished arterial

tension has the opposite characteristics, viz.: soft and compressible, implying deficient propulsive power or loss of tonus in the vessels. It is a pulse of weakness or debility. In fevers in general, a frequent, full, or small and soft pulse is the rule, especially after the first week, and during convalescence.

Dicrotism of the Pulse. In the healthy subject the finger applied to the radial artery is sensible of but one impulse, but it is possible to recognize one or two slight secondary elevations of the artery. These are the result of the recoil waves of blood, which, during the aortic systole, are thrown back on the semi-lunar valves, and thence rebound and travel outward to the periphery. In perfect health these waves can only be recognized by the sphygmograph, but if the tension of the artery is seriously lessened—as, for instance, in persistent high fever, 102°–104° F.—and if, further, the primary impulse produced by the systole is quick, short, but strong, the recoil waves may be felt by the finger. In individuals exhausted by chronic disease the arterial tension gives way even under moderate fever. In intermittent fevers dicrotism can often be noticed. If the pulse is very rapid, a double dicrotism may be observed.

Inequality of Pulsation. By this it is sometimes meant that the blood waves causing the pulse do not arrive at the two wrists at precisely the same moment, but in one radial it is delayed an appreciable interval. This is a symptom of aneurism of the arch of the aorta, the orifices of the innominate or subclavian artery being altered, and the pulse delayed in the arteries of the side toward which the aneurism lies. The radial pulses may not be alike in volume, indicating an aneurism, or the partial obstruction of the innominate or left subclavian, perhaps by a spicula of atheromatous deposit. It may be noticed in some cases of aneurism or aortic stenosis, that the pulse is noticeably postponed. Large aneurisms of

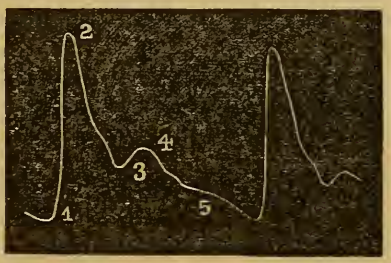
the descending aorta, in some cases, tend to retard the pulse wave, so that it is not perceptible in the femorals so soon as in the radials.

In inspiration the pulse-wave in all the arteries may be reduced in magnitude or suppressed, but returns to its normal volume or tension during expiration. A similar change occurs if any mechanical obstacle exist to the emptying of the left ventricle into the aorta; as, for instance, the adhesions of a fibrous pericarditis. Firm bands of connective tissue may enclose the great vessels and drag the aorta from its normal position, and attach it to the sternum. These bands are put on the stretch when the thorax is dilated on inspiration, and grasp or constrict the aorta, innominate artery, etc. The arterial pulse is rendered feeble, or even disappears, if the chest be expanded to its utmost; in expiration, on the contrary, it regains its normal calibre, and the pulse its normal volume. (Guttman.) Irregularity of pulsation also includes irregularities of volume and rhythm.

The sphygmograph is an instrument used to register the arterial tension, the pulse rate and its rhythm. The usefulness of the instrument in the physiological laboratory is indisputable, and in the clinical study of cases it affords facility in recording cases for reference. Its use is, however, auxiliary to physical examination of the arterial tension by auscultation or palpation, because the pressure at which the tracings are taken must be varied, and individual judgment must be relied on to assure one that the best record has been taken. Many instruments, like Pond's sphygmograph, in which the degree of pressure is shown by a graduated scale, do not represent the exact pressure on the artery, since the pressure is also brought to bear on the adjacent tissues, such as the tendons of the flexor carpi radialis, the radius, skin and fascia. In Pond's instrument the dicrotic wave is apt to be exaggerated by the recoil of the needle, owing to the weight of the ball

attached to it. The stiffness of a spring, which receives the first impulse of the artery, also militates against the accuracy of other instruments. Moreover, tracings taken from healthy persons vary within quite wide limits as to tension. Discounting these disadvantages, the instrument has a great value in recording cases for future reference and in presenting to the eye a study of the pulse, which, united to a careful analysis of the sum of the other clinical evidence, is of much diagnostic service.

In a normal tracing we have an unbroken line of ascent, or ventricular percussion systole, nearly vertical, but varying in height with the size of the vessel examined. Next we have the line of descent at an acute angle to the upstroke. This is due to the fact that the lever falls of its own weight; but it is caught, and lifted, partly by the tide of the onward blood current, partly by the portion of it which is sent backward toward the heart by the aortic recoil; this is called the tidal wave. The column of blood, after impinging on the already closed aortic valves, produces a new wave, which takes up the descending line, forming the second curve or dicrotic wave. After which we have the period of diastole, which consists in a wave springing from the dicrotic curve (called the aortic notch, since it indicates the closure of the semilunar segments) and a gradually sloping line, ending at the base line of the tracing. One of these curves, however, may be wanting. Besides these recoil waves, several other more or less marked undulations present themselves in the ascending or descending lines of the tracing, caused by oscillations



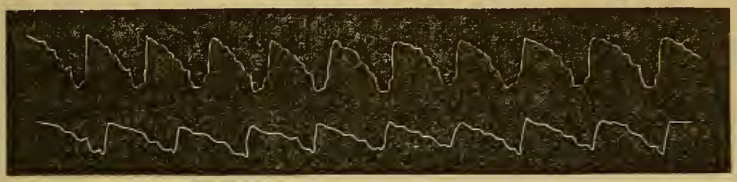
(AFTER FOSTER.)

NORMAL PULSE TRACING, MAGNIFIED.

1–2 Percussion up stroke.
1–2–3 " wave.
3–4–5 Tidal wave.
4–5 Dicrotic wave.
5 To base line, diastolic period.

of the arterial wall as it expands or returns to a state of rest, after the percussion impulse, especially if the systole is prolonged.

Tracings illustrate the following facts: The altitude and the approach to the vertical with an acute angle in the upstroke, is the measure of the force of the impulse and the consequent degree of arterial fullness, with the minimum arterial tension. The reverse is a percussion wave of diminished amplitude, deflected more or less from the vertical, with an apex appearing as a truncated cone. This tracing is an indication of the weakness of the systole, or imperfect arterial supply, or else a high degree of arterial tension. The line of descent is vertical, marked by dicrotic curves in proportion as arterial tension is diminished; the tidal wave is



(AFTER SANSOM.)

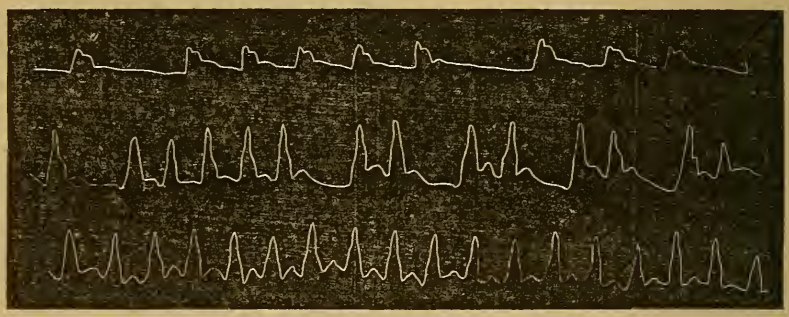
PULSE TRACINGS OF INCREASED ARTERIAL TENSION.

increased proportionately to the amount of blood jetted into the artery, or the duration of the ventricular systole, but is reduced in proportion as atheroma or reduced arterial tension prevails. If the summits of a series of percussion waves can each be touched by a parallel line, the volume of the pulses is equal. In all tracings the nearer the heart they are taken the shorter will be the primary wave, and the recoil waves will be more pronounced and nearer the summit of the wave of descent. One must be careful to secure a tracing while respiration is being naturally carried on, since on inspiration the intra-thoracic blood pressure is reduced, and the pulse wave is reduced in magnitude.

Pulse Tracings in Increased Arterial Tension are indicated by

a more or less marked deviation of the percussion stroke from the vertical; the summit is a blunt cone, instead of the normal acute angle; the first portion of the down stroke is more oblique than normal; the tidal or blood wave is increased; the dicrotic wave has a higher place on the tracing than is normal, or it may be but slightly marked.

Pulse Tracings in Low Arterial Tension are indicated in the up stroke by the increased height of the percussion wave, owing to the increased calibre of the artery. In the down stroke by the more vertical descent of the first portion of the trace, and by the decided prominence of the dicrotic wave, the aortic notch being lower than in the normal tracing. When the notch extends below the level of the base line, the pulse is said to be hyper-dicrotic.



(AFTER SANSOM.)

PULSE TRACINGS OF LOW TENSION, SHOWING IRREGULARITIES IN RHYTHM.

Pulse Tracings in Aortic Stenosis. The percussion stroke is more sloping, less ample, and more rounded, the increased force of the percussion stroke being neutralized by the stenosis. When there is less obstruction or simple roughening, the line of ascent is more vertical, its height is greater, and there is a more pronounced tidal wave, on account of the increased ventricular systole; a dicrotic wave is also present. If the stenosis is extreme, the recoil waves are not well marked, since the arteries are imperfectly filled.

Pulse Tracings in Aortic Regurgitation. The up stroke is ample and vertical, owing to the powerful impulse of the hypertrophied left ventricle, and the frequent presence of atheroma, indicating enlarged and rigid arterial channels. The recoil waves are enfeebled in proportion as the aortic valves become incompetent, and the column of blood flows back into the left ventricle, since the *point d'appui* of the blood column is removed. The down stroke is prolonged and vertical, and the dicrotic wave obliterated. The presence of a dicrotic wave indicates a partial functional persistence of valve structure. The dicrotic wave, if present, appears low down in the line of descent; slight increase of pressure on the artery will again obliterate it, since the recoil in these cases is always feeble. The diastolic portion of the trace is unusually flat and prolonged, indicating arterial emptiness during ventricular diastole. An attempt has been made to differentiate, by means of the sphygmograph, aortic regurgitation, due to associated degenerative arterial disease, from that of rheumatic endocarditis. The tracing in the former case is said to indicate greater arterial tension. In our experience aortic dilatation or actual atheroma, either primary or secondary to the ventricular hypertrophy, is so constant that this distinction is of little value. "In combined aortic stenosis and regurgitation the prolongation of the tidal wave is thought to be the measure of the stenosis" (*Mahomed*). The presence of some dicrotic wave naturally indicates that the valves are somewhat functionally efficient.

Pulse Tracings in Mitral Valvular Lesions. It is manifest that sphygmographic tracings of mitral regurgitation must be varied in relation with the amount of hypertrophy which is present. In mitral obstruction the arterial tension is apt to be low, and the percussion wave is abridged in amplitude, on account of the imperfect blood supply to the left ventricle, and the absence of hypertrophy of this portion of the heart.

The sphygmograph also records the frequency of the pulse. First, estimate the time the slide takes to travel; it is usually so constructed that six inches travel in fifteen seconds; to count the pulse, measure off six inches of the tracing, and multiply by four the number of pulsations observed.

The sphygmograph records the cardiac rhythm by the regularity of the succession of tracings both at the base as well as their summit. In interpreting these changes of cardiac rhythm, we must remember irregularity is both functional and organic.

For further details on this subject and sphygmography in general, *see* Dr. Mahomed's paper in the *Med. Times and Gazette*, for the year 1872. Also Dr. B. Foster's Clinical Medicine.

CHAPTER XII.

PROCESSES AFFECTING THE PERICARDIUM.

In lesions affecting the endocardium the predisposing causes of inflammatory processes are important aids to diagnosis. In pericardial disease a true estimate of the etiology is so fundamental as to be an integral part of each diagnosis. Pericarditis is almost never idiopathic; on the contrary, it is developed in direct association with rheumatism, with Bright's diseases, or as an inflammation propagated from an allied process in contiguous tissues. Pericarditis is sometimes a complication of pyæmia, scarlatina and other exanthematous diseases. The sources of inflammation likely to occasion pericarditis by contiguity are, inflammations of the pleura, especially of chronic type; inflammation in the mediastinum; abscesses beneath the abdominal diaphragmatic surface, developed by a peri-hepatitis; or chronic peritonitis. In a variously estimated proportion of cases both peri- and endocarditis exist. As in other serous membranes, the expression of the inflammatory process occurs in two forms, the acute, with effusion, either fibrinous or serous, at times mixed with blood, and the chronic, which mostly results in adhesions, which in turn may induce degenerations or dilatations of the heart.

Acute Pericarditis with Effusion. *Percussion.* One of the earliest evidences of pericardial effusion consists in the altered contour of the area of cardiac dullness. It is increased both transversely and vertically. In health the area of cardiac dullness in the adult scarcely exceeds three inches transversely at the fifth interspace. If in percussing from above downwards, an area of positive dullness exceeds this limit, or extends above the third rib, it becomes an evidence

of pericarditis. The outline of præcordial flatness is a blunt cone; the area of flatness may commence below, at the sixth or seventh rib, and possibly extend as high as the first or second rib. The transverse area of flatness may extend for an inch or more from the right border of the sternum, and to a variable distance beyond the plane of the left nipple. The transition from the outline of flatness to the outline of resonance is abrupt, just as in pleural effusions. The edge of the lung which normally overlaps the cardiac area is pushed aside, and the area of impaired resonance is abrogated. The percussion note is really flat, not dull, and the resistance to the fingers in percusssion is most perceptible.

The amount of a pericardial effusion varies, with comparatively short intervals of time, from day to day—almost from a morning to an evening advances or recessions of dullness can be defined. A very important aid is the change of the area of flatness when the position of the patient is changed from side to side, while at the same time auscultation or palpation will show an unchanged apex beat. Further, examination in the recumbent position will give a clearer percussion resonance, and the line of transition will be less abrupt as the lungs return to the position from which they were displaced by the fluid. Percussion may assure us that there is fluid in the pericardial sac; which may occur equally as a passive dropsy as well as an inflammatory effusion. Passive dropsy, however, occurs only in this situation together with other passive effusions.*

* There are serious objections to the deductions of the area of pericardial dullness in pericarditis from the results of artificial distention of the pericardium after death. The experiments of Dr. Rotch, of Boston, are as follows: When he injected a small amount of fluid into the sac, percussion gave an increase of præcordial flatness; beginning at the sixth rib, about two centimeters to the right of the sternum, it passed upwards in a curved line, with the convexity outwards, to the fourth right costal cartilage at its lower edge, then across the sternum to the upper border of the fourth

Palpation in Pericarditis. In pericardial effusion, both inflammatory or passive, the study of the apex beat shows that it is displaced or effaced. It is pushed upward and often outward to the left by the effusion, as the level of fluid ascends. When felt in the new position, the impulse is feeble, evidently distant and transmitted, and in some cases it is imperceptible. The position of the patient influences our study materially. If possible, incline the patient's person forward. This, if the manœuvre be admissible by the patient's condition, will render the apex beat more plain. Again, as the fluid increases or diminishes, the apex beat may become less distinct or more apparent.

Friction fremitus is a very valuable sign of pericarditis, although it is absent in a great many cases. The fremitus can be recognized if the examination is made in the early stages of acute pericarditis with moderate effusion, accompanying either the systole or diastole, or both; in the latter case the fremitus has a to-and-fro character. The fremitus occurs when the exudation is rich in fibrine, and the flakes are spread over the surface of the pericardium, especially near the base. At an autopsy the particles of fibrine appear like down or fur covering the heart. The friction of the heart's action masses these particles, and this condition has been compared on post-mortem to a cat's tongue. In order that these frictions may be recognized the amount of fluid must not be so great as to separate the heart from the chest wall, and pulsations must be sufficiently strong to cause a

left costal cartilage, and outwards and downwards to and to the outside of the nipple, passing down to the sixth or seventh rib. There was no vertical increase of flatness. When a large amount of fluid was introduced the area of flatness was increased upon similar outlines, viz: a much greater lateral increase from the upper border of the sixth rib to the lower portion of the third interspace, both to the right and left of the sternum, than above the third rib. The vertical increase of dullness was not materially increased.

definite rub. Pericardial fremitus or thrill, when it can be recognized by palpation, can also be detected by auscultation.

The pulse in pericarditis is only of value in connection with other signs. Dr. Walsh calls attention to the sudden variations in the pulse rate in pericarditis; a slight movement may increase the pulse from 60 to 90 or 140. Similar sudden rapid action of the heart is frequent from neurosal or emotional causes. The pulse may be irregular in force and rhythm, and may exist from the first before sufficient fluid is present to account for the phenomena.

Auscultation in Acute Pericarditis. Auscultation of the præcordia must be sedulously practiced in all cases in which etiological conditions suggest the possibility of pericarditis. In the primary stages of pericarditis, or throughout the history of cases in which the exudation is fibrinous, a rubbing or creaking sound may be audible, accompanying both movements of the heart. Sometimes the murmur is a soft, blowing sound, resembling the attrition of two surfaces of cloth, or the murmur may resemble so closely a valvular murmur that acoustically they are identical. The friction may occur rhythmically with one or the other sound of the heart, but much more commonly it is a to and-fro sound. Among the peculiarities of the pericardial friction sound are, that it is very transient. Walsh says that it may appear and disappear in six hours. It may disappear on account of the increase in effusion, to reappear and finally vanish gradually. Most commonly, however, friction sound is audible for several days. It is a very common incident for a friction sound to occur in a case in which an autopsy will reveal scarcely any lesion (pericarditis sicca). The characteristics of a pericardial friction sound are chiefly: 1st. Its change of location of maximum intensity, since when one mass of lymph may be polished smooth other roughnesses may

form. 2d. It is limited to the cardiac area; and is not propagated in the line of the transmission of valvular murmurs. 3d. It is synchronous with the movements, rather than the sounds of the heart. Commonly, it is heard over the base of the heart, or near the orifices of the vessels. 4th. It is a superficial sound; the valvular sounds are heard beneath. 5th. The change of posture may induce an increase of murmur, rendering a murmur which is faint in the vertical position quite distinct in the recumbent. 6th. The alterations common to the walls of the heart in endocardial disease are absent. This point will not always be of great value, since endocarditis is often associated.

When the serous effusion preponderates in a case of pericarditis the sounds of the heart, especially the first, are very faint and muffled, sometimes almost inaudible If the effusion is not too large the sounds are clear over the apex beat, which is usually displaced upward, and the sounds grow louder as the stethoscope is carried upward.

Inspection in Acute Pericarditis. The præcordia, especially in the young, may bulge, filling out the intercostal spaces. The apex beat is found upward and outward, perhaps into the fourth interspace. The impulse may be feeble, weak or fluttering. We notice, in some cases, considerable dyspnœa; in others none. Certain postures may give relief; for instance, if the recumbent posture on the left side is selected, the liver and heart both tend to exercise pressure on the pericardium, so that the posture on the right side is apt to be preferred.

General Symptoms of Acute Pericarditis. Pain is a delusive symptom of pericarditis, since an entire attack may transpire without a lament from the patient. Wild delirium may be an accompaniment of pericarditis, and the præcordia should be examined whenever the symptom complicates

rheumatism or Bright's diseases.* Associated gastric irritability is also, sometimes, a symptom liable to divert attention from its proper localization. We would again call attention to the frequent transitory character of the attack, and especially of that important symptom, friction sounds. These being produced sometimes by mere prominences of vascular tufts, with but insignificant exudation, may be relieved by treatment, and all signs vanish in a few hours.

Differential Diagnosis in Acute Pericarditis. In making the diagnosis of pericarditis, the etiology of the disease is of the first importance. One of the chief sources of error seems to be the possibility of mistaking a dilated, fatty heart, with feeble heart sounds, for the enlargement of the præcordia due to pericardial effusion. There are certain facts which separate the flatness of pericardial effusion from the dullness of an enlarged heart. The first of these relates to the change of præcordial contour in pericarditis, already noted. The line of transition from flatness into resonance is more sharp in pericarditis with effusion than in cardiac degeneration or dilatation. Respiratory percussion, on full inspiration, may be made available, if the patient is not too short of breath, to define more sharply the line of transition from resonance to flatness. In cases of simple enlargement of the heart, the area of cardiac dullness is diminished by the use of this method. Dilatation of the heart has a chronic history and is associated with venous stasis, while pericardial effusion begins suddenly and is secondary to other pathological processes.

* When delirium, with more or less stupor, occurs in acute rheumatism, the symptoms may be due to the elevated temperature, the so-called cerebral rheumatism. The temperature in these cases is always very high. In cases reported under this reference, temperature in a case of rheumatism suddenly rose to 108¾° F. Phila. *Medical Times*, May 30, 1874; Clinical Lecture on a case of Cerebral Rheumatism, so-called; use of cold baths. By H. C. Wood, M.D.

In differentiating pleurisy with effusion, note that the physical signs of the disease in the pulmonary apparatus can be noted in the back. In anterior sacculated pleurisy the position of the collection will best serve us when contrasting the symptoms.

Another source of error consists in a confusion of pericardial with pleuro-pericardial frictions. If a patient be desired to hold the breath, one may be certain that the sensation is not communicated from the lungs or pleura. But if the murmur is more pronounced at the end of full inspiration, it may be a pleuro-pericardial friction, caused by the grazing of the pericardial sac or aorta against the pleura.*

Pleuro-pericardial frictions have a fixed area of maximum intensity, not a variable one, as in pericarditis, although the intensity of the sound varies in pericardial more than in exo-pericardial murmurs. Finally, if the results of other physical signs favor pleurisy, the friction may have a pleural origin. Exo-cardial murmurs may exist indefinitely without damage to a patient, and their diagnosis occurs mostly as a refinement of skill in chronic cases.

Pericarditis may be combined with endocarditis. However much the pericardial murmurs resemble the endocardial, the latter can be recognized by the depth of the plane at which they are developed, by their transmission in the route of the blood current, and by their being in association with definite lesions of the heart muscle. The respective dates of the commencement of the endo- or pericardial disease must be determined chiefly by the history.

The Terminations of Acute Pericarditis. The issue of a case of pericarditis may be summed up as follows: If death

* Walsh refers to a friction produced by the pulsation of the aorta, which is diastolic in rhythm, or an exo-cardial friction between the aorta and the pleura. The diastolic rhythm and increase during full inspiration may help to establish their identity.

occurs, it is on account of the amount of pericardial effusion, especially if pericarditis complicates the condition of a heart already weakened by endocardial disease or fatty degeneration. In Bright's diseases the prognosis is also unfavorable. High temperature is a serious symptom, but in a thoroughly healthy subject, in rheumatic pericarditis, the prognosis is not unfavorable.

Chronic Pericarditis. When a patient comes into one's presence with chronic heart disease, one must always ascertain if the symptoms be really closely connected with the lesions detected by physical diagnosis. In all our studies of the heart a strict censorship must be maintained, to obviate a disposition, on the one hand, to disregard the complaints of a patient, through an enthusiasm over the physical signs of disease, or else, in chronic cardiac disease, to attribute all the complaints more or less directly to the heart. In no condition of chronic heart disease is the above caution more applicable than in chronic pericarditis, since it may produce many serious results, or the lesion may be devoid of inconvenience. The lesions of chronic pericarditis are very similar to those of chronic pleurisy. 1st. The effusion in acute pleurisy may remain without tendency to increase or diminution. 2d. Adhesion or agglutination of the pericardial surfaces may occur. This latter lesion induces either cardiac hypertrophy and dilatation, or cardiac atrophy. 3d. Retained pericardial effusion may become purulent. The first class of lesions require no elaboration, since the signs of effusion are always the same. The second condition is one of the hard problems of physical diagnosis. Again, at the outset, we require an unequivocal history. We believe that dyspnœa, of the form peculiar to the heart (viz. : increased by exertion), is a sign of chronic pericarditis with adhesions, provided that careful study excludes any other form of cardiac or pulmonary disease, or any neurosal disturbance of the

heart. In hypertrophy connected with pericardial adhesion, the heart's apex may pulsate as high as natural, in spite of the enlargement; or the heart may be drawn upward and outward, and to the left. This is not the history of hypertrophy and dilatation without adhesion. If the pericardial surfaces are extensively united, the præcordiæ may be depressed and the interspaces drawn inward during the systole. The heart's apex may be drawn upward and outward; even the lower portion of the sternum may be contracted. The limits of cardiac dullness may be enlarged and unchangeable in varying postures of the body, or in full inspiration and expiration.

There may be no change in the rhythm of the heart, or there may be a very great alteration of rhythm, including irregularity. Walsh thinks that the systolic dimpling of the interspaces only happens if there be, in addition to pericardial adhesions, pleuritic adhesions in front of the organ, or if the agglutination of the pericardium be combined with cardiac hypertrophy. We would refer, in this connection, to what was said, under the head of Emphysema, in reference to the loss of resiliency of the distended chest, and the liability to depression of the interspaces during inspiration. We have also seen the dimpling of the interspaces and wave-like vermicular tremor of the præcordial spaces, in pericardial adhesions, without pleuritic adhesions, and in individuals whose chest walls were much emaciated and the heart enlarged and dilated but without pericardial adhesions. We may fairly say that the diagnosis of chronic pericarditis can only be effected by taking into the fullest consideration the predisposing etiological causes and the sum of the physical signs just laid down, together with a general diagnosis by exclusion.

Paracentesis of the Pericardium. In pericarditis with large effusion paracentesis may be demanded. The point of election, according to Roberts, in his monograph on this subject,

is in the fifth interspace and situated from two to two and a quarter inches to the left of the median line of the sternum. Aside from injuring the heart or puncturing the pleura, the selection of this point enables the operator to avoid injuring the internal mammary artery. "This artery runs parallel with the edge of the sternum until it reaches the lower edge of the sixth costal cartilage; here or in this neighborhood it bifurcates into the superior epigastric, continuing directly downward; and the muscular phrenic, which runs downward and outward in the sixth interspace. The trunk in this region is somewhat less than three millimeters in diameter, and its two branches from one and a half to two millimeters. Cruveilhier and Sappey give as the average distance from the sternum four or five millimeters. Some measurements of Roberts show it to be from one-quarter to one-half an inch, which would correspond to about six to twelve millimeters, from the left border of the sternum."

TABLES OF REFERENCE.

(Taken from Peacock's Croonian Lectures for 1865).

Weight of Healthy Heart in Males, . . 9 oz.

Weight of Healthy Heart in Females, . . 8 oz.

DIMENSIONS OF THE HEALTHY HEART IN ADULT MALES AND FEMALES.

	BIZOT.		PEACOCK.	
	Males. Eng. inches.	Females. Eng. inches.	Males. Eng. inches.	Females. Eng. inches.
Circumference of Heart, ...	. . .	. . .	9.209	9.236
Girth of Right Ventricle, ...	. . .	. . .	4.919	5.184
" Left "	. . .	. . .	4.289	4.049
Length of the Cavity of the Right Ventricle, . .	3.3	3.01	3.821	3.925
" " " Left " . . .	3.05	2.75	3.333	3.197
Thickness of Walls of Right Ventricle, Base, . .	.168	.142	.164	.164
" " " " Mid-point, .	.124	.114	.176	.177
" " " " Apex, . .	.091	.082	.125	.118
" " Left " Base, . .	.399	.381	.452	.432
" " " " Mid-point, .	.457	.426	.532	.497
" " " " Apex, . .	328	.301	.214	.222
" Septum, ...	.435	.39	.51	.421
Circumference of Right Auriculo-Ventricular Aperture, .	6.812	4.184	4.74	4.562
" Left " " " .	4.04	3.67	4.	3.996
" Pulmonic " " " .	2.871	2.671	3.552	3.493
" Aortic " " " .	2.754	2.557	3.146	3.019

"The girth was ascertained, generally, immediately the heart was removed from the body; the dimensions being taken, by a piece of string or tape, from the anterior branch of the coronary artery in front to the large vein behind, and passed over to the broadest part of the organ. The cavities were laid open by incisions, made about half an inch on each side of the septum; that into the right ventricle following the curve of the septum, that into the left passing direct from the base to the apex. The length of the right ventricle was measured by string passing along the convexity of the septum from the base to the apex; that of the left ventricle was taken in a direct line, from the base or upper margin of the muscular septum, to the apex. The thickness of the walls was ascertained on each side at about half an inch from the base, at the mid-point, and at a quarter of an inch above the apex. The septum was cut across midway between the base and the apex, and the width measured at the mid-point of the section so produced. The dimensions of the orifices were ascertained by balls of different diameters."

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